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HEMATOLOGY.

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INFECTIOUS MONONUCLEOSIS.

This condition was first described by Pfeiffer, of Vienna, in 1889 under the name of glandular fever. The disease received little publicity for many years, even during World War I, when many cases doubtless occurred but were not reported. Since the publication by Sprunt and Evans of another account of the disease in 1920, under the name Infectious Mononucleosis, numerous reports of isolated and epidemic cases, including some with bizarre complications, have appeared in the literature.

The disease is clearly infectious, but the epidemiology is obscure. The infecting agent is a virus, and the disease has been transmitted in monkeys by an agent obtained from bacteria-free nasal washings.[1] The exact mode of transmission is not known, but it seems to be by droplet infection similar to the common cold. Cases may occur sporadically or in epidemic form. The virus is highly infectious, though it resembles other viruses in that inapparent infection is common. This may explain why some persons, exposed to the

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infection, do not contract the disease.[2] The infective period is unknown. One attack probably confers a lasting immunity, and reported recurrences are probably due to a similar condition of infectious lymphocytosis, or other infections, or to relapses of the original infection. It occurs in all ages, but is most common in children.

The incubation period is approximately five to 15 days. The milder cases can be divided into three groups according to the classification of Tidy:[3] The glandular fever type occurs in children and is characterized by enlargement of the cervical glands and little inflammation of the throat. The angiose type occurs in young adults and is characterized by the development of a true membrane on the throat, with enlargement and tenderness of the cervical glands. There is fever out of proportion to the patient's appearance. The membrane separates within a week, and the patient rapidly improves. The adult type is more insidious, often preceded by a longer incubation period and accompanied by less glandular enlargement and inflammation of the throat. The prolonged fever is most characteristic, but it is difficult to distinguish from other causes of prolonged fever. There is lymph node enlargement, most frequently in the postcervical chain, and this may be tender. The spleen is palpable in about half the cases.

Severe cases have been reported complicated by splenic rupture,[4] encephalitis and other neurological disorders,[5] hepatitis, with and without jaundice,[6] and thrombocytopenic purpura.[7] The disease may resemble the acute surgical emergencies of the abdomen, poliomyelitis, Guillian-Barré syndrome, or other neurological infections, hepatitis, or the many syndromes associated with thrombocytopenic purpura, according to the complication present. These widespread manifestations have been explained by the generalized involvement of the cases which have come to autopsy. Focal infiltrations of mononuclear cells in a number of organs, including the liver, heart, kidneys, lungs, spleen, testicles and brain, have been observed.[8]

Diagnosis is made from the clinical course when it is typical, and from the blood when it is not. In the milder forms,

there may be an initial leucocytosis, from 15,000 to 20,000 cells, with 75 per cent polys. This finding may be entirely misleading if another count is not done. Usually there is a leucopenia of the polymorphonuclear cells from the onset. The abnormal lymphocytes characteristic of the disease then appear, with round or bean-shaped nuclei, usually without nucleoli, and with abundant "foamy" cytoplasm which may be folded on itself. These cells are not confined to infectious mononucleosis alone, but are found in a number of other infectious diseases. There is then a lymphocytosis for a variable length of time. Difficulties arise in the differentiation from acute leucemia, which usually shows more young forms in the blood and more toxicity, and chronic lymphatic leucemia, the early stages of which closely mimic infectious mononucleosis. The heterophile agglutination test of Paul and Bunnel is helpful in making the diagnosis in all but the most obvious cases. A significant titer varies with the method employed, so will vary from place to place. A positive reaction may occur very early, as in the milder cases, or it may not appear later with subsidence of the constitutional symptoms, so it is best in suspected cases to repeat the test where it is negative when first done. A positive test is considered diagnostic, but a negative test does not preclude the diagnosis, since about 10 per cent of these cases fail to demonstrate antibodies.

The course of the disease is usually benign. Treatment is directed at the complications, since there is no effective treatment of the primary disease. Perhaps gamma globulin, especially that from a convalescent patient (vide infra) may be of benefit in attenuating or preventing the disease.

Our experience with aureomycin has been too limited to warrant a definite statement, but in the few cases so treated the angina and the cervical adenitis receded rapidly.

THROMBOCYTOPENIA.

The conditions likely to be associated with a reduction in the platelets are the exanthemata and drug sensitivity. Scarlet fever,[9] German measles[10] and chicken pox[11] have recently

been reported in association with thrombocytopenia, prompt recovery usually taking place coincident with recovery from the exanthemata. It has also been reported following the administration of potassium iodide,[12] quinidine[13] and associated with pancytopenia following the administration of tridione, a new anticonvulsive.[14] The presence of idiopathic thrombycytopenia must always be suspected in such cases, even when a potentially dangerous drug has been administered. The idiopathic cases have the characteristic immature, nonproductive megakaryocytes in the marrow in abundance, as compared to those produced by drugs which have none. Treatment consists in removal of the causative agent, if one is known, and administration of methionine, or foods rich in this amino acid, B complex vitamins, including folic acid, and fresh transfusions.

GRANULOCYTOPENIA.

The occurrence of bone marrow hypoplasia associated with the drugs commonly used is being reported with alarming frequency and should be kept in mind. Following its widespread use in the war,[15] atabrine has been implicated, causing aplastic anemia. Tridione, a benzene derivative, has proven to be a bone marrow depressant in some patients, causing thrombocytopenia, as noted above, and granulocytopenia. This effect comes on insidiously without relation to size or duration of dosage and is corrected when the dose is discontinued. The white count of such patients should be followed at regular intervals. It is a well known fact that thiouracil and the sulfonamides can cause depression of the white count, especially the granulocytes. The same treatment as for thrombocytopenia should be given; pentanucleotides have also been used with equivocal results.

BLOOD DERIVATIVES.

The vast program of red cell preservation and plasma fractionation carried on by Cohn, *et al.*, since 1941 for the National Research Council and other agencies has resulted in a much better understanding of the contents and functions of

the blood. In addition, many useful products have been made available. An acid dextrose citrate solution has been found which will preserve 70 to 80 per cent of the red cells for 21 days, if stored at a temperature between 4 to 10°.[16] Plasma and albumen have been used to increase the circulating protein in conditions where it is decreased, such as in shock and in the preparation of debilitated patients for surgery.

For the otolaryngologists, the most important derivatives are those which assist coagulation. Fibrin foam is a light, absorbable material which when used with thrombin has proven effective in a variety of surgical conditions. It deserves a try in cases of epistaxis resistant to routine cautery and packing.[17] Fibrin film, containing thrombin, was first used as a dural substitute but has recently been employed to repair perforations of the drum and nasal septum.[18] It is pliable and can easily be molded into a suitable shape. In suspected or frankly infected areas, these agents are equally effective with the sulfonamides and penicillin. Recently, "gelfoam," absorbable gelatin sponge,[19] and "oxycel," oxidized cellulose,[20] have received clinical trial, especially in nasal packing for epistaxis. They seem to be as effective as the blood derivatives, and together with them have the advantage over routine packing, in that they are absorbable and do not have to be removed. Thrombin has been used in nasal surgery to facilitate the attachment of skin flaps[21] and together with fibrinogen has been used in plastic surgery.[22] Fraction I of Cohn contains the specific antihemophilia globulin "thromboplastinogen," and 200 to 600 mg. of this given intravenously will temporarily restore the clotting mechanism of a hemophiliac to normal. This will allow him to undergo minor surgery without complication from hemorrhage and stops bleeding when it occurs following an accident.[23]

The gamma globulins present in Fraction II contain the antibodies circulating in the plasma. These can attenuate or prevent epidemic hepatitis[25] or measles, depending on how early they are given.[24] The antibodies for mumps are present in very low titer in normal plasma but are present in much

greater quantity in the plasma of convalescent patients. Such convalescent globulin will markedly reduce the occurrence of orchitis in adolescent and adult males with mumps, and if given early enough may have a beneficial effect on the encephalitis that occasionally complicates mumps, with its resultant deafness.[26] Similar convalescent gamma globulin is available for the prevention and treatment of pertussis.

A serious disadvantage to the use of plasma fractions is the frequency of homologous serum jaundice following their use. The pooling of plasma prior to fractionation permits a thorough mixing of the infected plasma with the rest. The great resistance of the causative virus permits it to remain viable during the process of fractionation. Measures have been developed for the inactivation of the virus during fractionation, the most prominent being ultraviolet light.[27]

THE CHEMOTHERAPY OF LEUCEMIA AND LYMPHOMA.

NITROGEN MUSTARD.

The poisonous mustard gas used in World War I exerted its harmful effect by its surface vesicant action, although at that time it was known to cause leucopena.[28] The renewed interest in poison gas in this war was centered around mustard gas and several of its nitrogenous analogues. They were found to have cytotoxic action on many issues when given internally. This action was unique for a chemical agent and resembled X-ray more closely than anything else.

The mode of action of these drugs is probably the inactivation of intracellular enzyme systems by the alkalinization of their essential carobxyl, sulfahydroxyl and amino groups.[29] There is no indication that these compounds are specific for malignant tissue; however, since in smaller doses they prevent mitoses all over the body, their action is most marked in the rapidly dividing malignant tissues. Given in larger doses, they cause nuclear fragmentation and changes in the cytoplasm, and perhaps this mechanism is responsible for their clinical usefulness.

These drugs have been given a thorough clinical trial on patients with malignant disease. Encouraging results have been obtained in Hodgkin's disease, lymphosarcoma, chronic myelocytic and chronic lymphatic leucemia and polycythemia vera.[30-34] The reduction in pain and fever following treatment has been especially promising; in some cases, almost immediate. Often there is reduction in the size of the spleen and lymph nodes and a return of the feeling of well-being. Solitary lesions are probably best treated by X-ray, mustard being reserved for the more generalized variety and for those resistant to X-ray, since they will occasionally undergo a satisfactory response to mustard when they will no longer respond to radiation. There is no evidence to indicate that mustard is superior to well administered X-ray, and it is probably a matter of convenience which form of treatment is used. As with X-ray, the remissions induced depend on the disease being treated. Nitrogen mustard has little beneficial effect in the acute leucemias, and since it may depress hemoglobin formation, it is not recommended.

The most effective compound tested to date has been methyl bis (B chlor ethyl) amine hydrochloride. The dose is 0.1 mg./kg. of body weight intravenously in saline for three to six consecutive days, depending on the white count. Care must be taken to see that none of the solution enters the tissues around the vein because of its irritating effect. Nausea and vomiting usually follow the first few treatments. The amount of drug given is usually limited by the white count since it tends to fall, especially in polys; there may also be a depression in the platelets. In those patients who respond, there is usually a rise in the red count if it was low before treatment. Recovery from the depressant effect on the blood is usually complete in four to six weeks, at which time a second series of treatment may be given if necessary. Just as with X-ray, a lesion which has responded well the first time may fail to do so, or may respond only slightly the next time.

URETHANE.

In 1946, Haddon and Sexton reported the inhibitory effect of the common laboratory anesthetic, urethane (ethyl car-

bamate), on rat cancer.[35] For this reason it was tried on human cancer with little success. A leucopenia was observed in these patients during treatment. It was, therefore, given to patients with leucemia and found to have a favorable effect on one-third, causing a reduction in the size of the lymph nodes and spleen, and reversal of the blood picture toward more normal values.[36] It has the advantage of being almost nontoxic, and can be given by mouth. The dose is 3 to 4 gms. per day for 42 days, and a maintenance dose of 0.5 to 1 Gm. usually sustains this effect.

FOLIC ACID ANTAGONISTS.

The widespread and fundamental importance of folic acid in hematopoiesis is apparent from its action upon all the elements of the marrow.[37] Farber, *et al.*, noticed an "acceleration phenomenon" in the leucemic process in children with acute leucemia following the injection of folic acid conjugates.[38] It was, therefore, supposed that some antagonists of folic acid could be synthesized, which would block this action of folic acid in leucemic patients and restore the blood toward normal. Such compounds have been developed and are now being tested, the most promising being aminopterin.[39]

RADIOACTIVE ISOTOPES.

Many of the elements have been prepared in isotopic form for the treatment of neoplastic disease. The purpose in doing this is to selectively deposit radioactive material in the disease area to exert its destructive effect locally. The difficulty is finding a material which can be concentrated sufficiently in the tissue to be treated. The most promising is radioactive phosphorus in the treatment of polycythemia vera, but this does not seem to be superior to nitrogen mustard.[40] There is promise in the use of these radioactive elements, but much better ones need to be developed.

HEMORRHAGIC DISORDERS.

The great amount of investigative work done on the clotting mechanism has added to the understanding of this process,

but has not changed any of the basic elements as outlined by Howell in 1935.[41] The recent excellent paper by Quick, Honarato and Stefanini outlines the coagulative process as follows:[42]

1. Thromboplastinogen platelet enzyme: thromboplastin. The activated thromboplastin reacts immediately:
2. Thromboplastin + prothrombin + calcium = thrombin.
3. Fibrinogen thrombin: fibrin.

The main difference with the older theory is concerned with thromboplastin. It is now thought to come from the plasma, not the platelets, but requires an enzyme from the platelets to convert it to an active form.

In hemophilia, the thromboplastin precursor in the plasma is almost absent, but when this is supplied, as by fresh plasma or the Fraction I of Cohn, the blood of hemophiliacs clots readily and well. To understand hemophilia, it must be understood that a small amount of thrombin, formed from only a very small amount of thromboplastin precursor and prothrombin, is enough to initiate coagulation. This explains why hemophiliacs can have a normal or nearly normal clotting time as done by the most accurate methods[42] and yet suffer from repeated hemorrhages. Since they have only a small amount of thromboplastin precursor, they form only a small amount of thrombin, which can convert only a small amount of fibrinogen to fibrin. This small amount of fibrin may be enough to clot blood in a test tube, but not enough to occlude a severed blood vessel; hence, the patient may have a normal clotting time and yet not be able to clot blood in a severed vessel, especially if it is a larger one. This demonstrates the unreliability of the clotting time in hemophilia. It is of value only when it is normal, or only slightly elevated, since such patients can be predicted to have only slight bleeding. The amount of prothrombin remaining in the plasma after clotting time is a more reliable test of the coagulative defect in hemophilia, since very little is removed. Normally most is removed during coagulation.

The prothrombin time as measured by the method of Quick is a chemical determination of the prothrombin activity. It is important to remember that prothrombin combines with thromboplastin and that if prothrombin is deficient, clotting will not occur or will be defective. The hemorrhagic danger level is indicated by a prothrombin time of 25 seconds, which corresponds to a prothrombin activity of 20 per cent. Dicoumarol, the toxic agent found by Link, *et al.*,[43] to be responsible for the hemorrhagic disease developing in cattle from eating spoiled clover hay, interferes with the production of prothrombin in the liver. It has no action on the clotting mechanism directly. The action on the prothrombin time is delayed 48 hours after the drug is started while the circulating prothrombin is removed from the blood. This action is opposed by certain naphthoquinones, the so-called vitamins K,[44] which are useful in combating overdose with dicoumarol. Heparin is the name given to a group of substances having an antithrombin action. It is given in doses of 50 to 100 units intravenously to adults, acting immediately to prolong the clotting time. Its action is brief and inconstant, because of excretion and inactivation, and the dose must be repeated every three hours. By giving it subcutaneously in a suitable menstrum the absorption can be delayed and only one dose per day is required. Dicoumarol is given in doses of 200 to 300 mg. by mouth, providing the prothrombin time is normal before treatment is begun. Later, doses are given according to the prothrombin time, which should be maintained between 20 to 35 per cent. In venous sinus and jugular vein thrombosis, when anticoagulant therapy is indicated, heparin and dicoumarol are started together. The heparin is continued until the dicoumarol begins to have its effect, usually at the end of 48 hours.

Dicoumarol is similar to the salicylates, and can be synthesized from them. The latter have been found to have a weak antiprothrombin action, especially aspirin.[45] There are numerous conflicting reports in the literature of the frequency of late tonsillar hemorrhage due to the salicylates.[46,47] The final answer has not been found. If the salicylates have any rôle in the production of late tonsillar hemorrhage, this diffi-

culty can easily be prevented by the administration of vitamin K. The usual dose is 5 to 10 mg. by mouth or parenterally.

The most perplexing and important disorders are those due to the capillaries. Vitamin C deficiency probably has very little importance in tonsillar hemorrhage, as is supposed by some.[48] Rutin, a derivative of flavone and closely related to hesperdin (vitamin P), citrin and ascorbic acid, is obtained from buckwheat leaves, flowers, tobacco, rue herb and many other plants. It has been shown to reduce capillary permeability when artificially induced in animals,[49] and in man in a variety of pathologic conditions.[50] It has caused a halt in the progress of retinal changes in patients with diabetic retinopathy, but has not corrected any of the existing damage.[51] It has also been of benefit in the treatment of epistaxis in some patients with hereditary hemorrhagic telangiectasis.[52] It seems worthwhile to try it, in doses of 50 to 100 mg. per day, with 100 to 200 mg. of vitamin C in capillary disorders with hemorrhage.

FOLIC ACID AND VITAMIN B_{12}.

The name Folic Acid was given by Mitchell, *et al.*, to an extract of spinach found to be necessary for the growth of str. fecalis.[52] It is now used to refer to the group of pteryolglutamic acids with similar hematopoietic activity found in nature and synthesized. This extract of spinach is essentially similar to the L. casei factor obtained from yeast,[53-55] the vitamin B extracted from liver necessary for the prevention of pancytopenia and retarded growth in chicks,[53-55] and the vitamin M necessary to prevent a sprue-like syndrome with pancytopenia in monkeys fed a synthetic diet containing all the known essential nutritive elements. It has been synthesized by Waller, *et al.*,[56] who found it to contain para amino benzoic acid, glutamic acid, and a pterin nucleus related to the yellow pigment found in butterfly wings.

The daily requirements of this vitamin have not been determined, but the amount present in the normal diet is much less than the amount required therapeutically. A great many bacteria produce this vitamin, and it may be that part of the

human requirement is derived thus.[57] There are no known deficiency states of folic acid, but the important symptoms that can be relieved by treatment with it are pancytopenia, retarded growth and gastrointestinal disturbances. Folic acid has proven effective in the treatment of the hematopoietic phase of Addisonian pernicious anemia, nutritional macrocytic anemia and neutropenia, and the macrocytic anemia of infancy and pregnancy, and following gastric resection.[58] There are no anemias responsive to folic acid that do not respond to liver extract. The action of liver extract in Addisonian pernicious anemia is to convert the conjugated form of folic acid, as it appears in the diet, into the active free form. Thus free folic acid when given directly does not need liver extract to be effective.[58] Folic acid does not reverse or prevent the neurological manifestations of Addisonian pernicious anemia, and may even precipitate their appearance in patients showing a good hematological response.[59] Other experimental studies also indicate a general fundamental rôle of folic acid in the human in addition to its effect on hematopoiesis.[60] It has been shown to increase the resistance of animals to strep and influenza infection as compared to animals not receiving it.[61] The results in treating aplastic anemia have been too variable to be conclusive, but folic acid is not curative. Thus pernicious anemia seems to be a multiple deficiency disease. Folic acid is capable of correcting the hematopoietic but not the neurological deficiencies. For this reason "the use of folic acid alone in the treatment of these cases is at present not recommended."[58] The usual therapeutic dose is 10 mg. per day by mouth, and 5 to 10 mg. per day as a maintenance dose.[58] If the disease doesn't respond in two weeks, it is probably not due to a folic acid deficiency. There is reason to believe that the larger doses, while not being more beneficial, may even precipitate the appearance of neurological disturbances.

The isolation from liver extract of a very potent antipernicious anemia factor, vitamin B_{12}, was carried out by Rickes, *et al.*[62] It was demonstrated by Shorb to promote the growth of L. casei.[63] This pure crystalline product seems to be the

long sought active principal of liver extract. It is active when taken orally alone, more active when combined with gastric juice, and most active when taken parenterally, in each instance resembling liver extract.[64] Doses as small as 5 to 25 micrograms have caused a clinical and hematological response in two cases of pernicious anemia, two cases of nutritional macrocytic anemia, and one case of nontropical sprue.[65] More recently, responses have been reported in cases of tropical sprue[66] and combined system disease.[67] This last patient was allergic to beef and hog extracts but had no untoward reaction to treatments with vitamin B_{12}. The advantages of potency, effectiveness against all the manifestations of the disease, ease of administration and absence of allergic properties make vitamin B_{12} the drug of choice in the treatment of macrocytic anemia. Until it becomes available, it is best to use a potent liver extract, with or without folic acid.

SUMMARY.

The advances in hematology of interest to the otolaryngologists in the last four years are presented. The epidemics of infectious mononucleosis during and since the war have increased the importance of this disease, so a brief review of it is presented. The blood fractions have been made available for assisting coagulation, preventing or attenuating infection and many other uses. Chemical agents are now available for the treatment of leucemia and lymphoma, the most important of which is nitrogen mustard. Two new products have been obtained from spinach and liver, folic acid and vitamin B_{12}, respectively, which are very powerful in the treatment of macrocytic anemia and related conditions. In addition, there have been advances in the understanding of the coagulative process and the diseases associated with it. The implication of many of the commonly used drugs in the development of hypoplasia of the marrow in varying degree makes it important that the potential danger of these drugs be understood.

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PSYCHOSOMATIC ASPECTS OF DISEASES OF THE EAR, NOSE AND THROAT.*

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INTRODUCTION.

The interrelationship between emotional experiences and physiologic processes is no longer a speculative one. This relationship is now accepted as a tangible and concrete factor in the perception of disease. The influence of emotional variations on body functions has been recognized and studied with interest in every phase of medicine. The alteration of clinical symptomatology by psychic factors has been repeatedly witnessed by us as otolaryngologists. To underestimate the importance of these psychosomatic factors in otolaryngology is to do a great injustice to our patients and ourselves as surgeons.

A psychic and somatic relationship may infer a feeling of separation. Rather do I mean to identify each as a stratification of the other. This, then, converts the psyche and the soma from a dissociated duality into a dominant unity.

It is my purpose to present some of the aspects of this body-mind unity as applied to practical otolaryngology and particularly to show how they affect medical and surgical results.

In accepting this orientation it might be wise to re-examine one fundamental question: namely, what is illness? If we are agreed that illness is a phase during which an individual is

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unable to be serviceable to himself, to his family, his community and to his earning occupation, then the cause of that illness must be our chief concern, whether its origin is psychic, organic or both. The patient's serviceability is limited, whether his illness is due to a fractured leg or to an acute anxiety. Hostilities, insecurities and anxieties are just as significant manifestations of pathology as are pain and tenderness. The responsibility of the physician, therefore, is to re-establish this lost serviceability and to rehabilitate the patient organically and psychically.

How do we as otologists fit into the complex structure of Psychosomatic Medicine? I should say that our responsibility is twofold: First, we must devote ourselves to eliminating, in our young patients, those traumatic experiences which may lead to adult neuroticisms. Second, we must re-evaluate our concept of organic disease and interpret it in terms of the personality.

I have arbitrarily chosen a discussion of tonsillectomy to emphasize the contribution we can make in avoiding psychic trauma in childhood. Tonsillectomy is by far the most common single operative experience of childhood. So common in its universality, in fact, that we have ceased to recognize its portent and effect on the future destiny of the child. In most cases, tonsillectomy represents the first admission of the child to a hospital and in most instances is the first painful separation of the child from his home and security. Let us for a moment trace the usual preoperative and postoperative story.

The child is brought to the office of the otolaryngologist, who sincerely tries to develop a pleasant relationship with his patient. Tonsillectomy is advised, the date is arranged and the experience is set. Except in those few cases where parents are attuned to the importance of careful psychological preparation, a great injustice is done to the child. A composite story reveals that the child is told that on a certain day he can anticipate a pleasant and new experience. Instead of that,

he finds himself on that day suddenly confined, by a "clever" ruse, to a hospital room far removed from the scene of his anticipated joy. Strange, serious, white-robed people are milling about him. He is almost immediately undressed, emphasizing his "capture." It is difficult to comprehend the impact of such terror and confusion, and to evaluate its disastrous sequelae. There is, first, the immediate resentment at having been lied to and tricked into this situation. Then there is the distrust of the child for the parent, which distrust may in one moment negate all the protection and support previously given. Unfortunately the parents are least equipped at this time to give solace and comfort, and usually cannot help but transmit their own apprehension and concern to the child. Without warning, one or two persons, the operating room attendants or nurses, gentle and kind, but at best strangers, pick up the struggling child and whisk him away to an unknown destiny.

Can we project ourselves into the terrifying moment when first the child enters the strange, white-walled, instrument-filled operating room? Before he can accommodate himself to this picture, a new terror descends upon him . . . for he is now overpowered and restrained, anesthesia begun. Do we who have witnessed this struggle adequately appraise the profound effect which this moment of terror has upon the impressionable child? He leaves his conscious state aware that his parents have deserted him. There is a shattering loss of faith in parental protection and complete distrust of their motives. The struggle between the conscious and the unconscious during the early stages of anesthesia is tantamount to dying. It is ironic that at this very moment the orderly usually feels personally insulted when the child refuses to take it lying down!

The immediate or delayed reaction to this relatively simple surgical procedure may be alterations in behavior, family antagonisms, negativisms, night terrors, hostilities, phobias, obsessions and other psychoneurotic patterns. For us, a most important consequence may be the lifelong fear and distrust of doctors, hospitals, anesthesia and surgery. At best, even

with the most careful preparation, this experience is far from a pleasant one; but we can attenuate the trauma by the proper handling of the child and the parent. Many of us feel that our chief obligation is to remove the tonsils, and that the future happiness of these children is not within our surgical province. Yet, that responsibility is more distinctly ours as we become aware of our important position in the psychosomatic approach.

I should like to suggest a few steps that might be taken to obviate the psychological pitfalls of tonsillectomy:

1. Give the child a forthright and honest explanation of the reasons for the operation.

2. Try to avoid the element of surprise and confusion at the hospital. A recognizable picture of the hospital, the operating room, white-gowned nurses and doctors should be described.

3. Explain that the purpose of anesthesia is to spare the child pain during the operation. Assurance must be given that this forced sleep is temporary and will be followed by complete awakening and survival. The ideal anesthetic is one chosen for surgical efficiency and psychic consideration.

4. Emphasize to the parents the need for constant reassurance and affection in the postoperative period.

5. Whenever possible, the doctor personally should lead the child from the mother to the operating room. The previously established child-doctor rapport here pays dividends in the sustained security.

6. Permit overt manifestation of postoperative hostility, rather than suppress it. The psychic damage of such suppressions is fraught with subsequent danger.

This realistic portrayal of the emotional aspects of a tonsillectomy is not meant to be pessimistic or foreboding. Nor is it meant to condemn a most efficacious and health restoring procedure. I mean only to illustrate that adult neuroticisms

may be traced to such experiences. Often it remains for the expert, the psychoanalyst, to uncover such a basis for fears, rejection and insecurities.

Let me re-emphasize that I do not mean to negate the value of a tonsillectomy because of potential psychic trauma. Rather do I want to consider psychic trauma as another pre- or post-operative problem to be avoided as we would hemorrhage or infection. By our very awareness of its potentialities we may make an intrinsically unpleasant experience less so, psychically and somatically. This, too, is a great surgical contribution.

I should like now to devote myself to the second of the two classifications, the interpretation of organic disease in terms of the personality.

The following subgrouping is a useful though superficial one for psychosomatic identification:

Group One consists of — The estimated one-third of all patients who do not have any definite bodily disease to account for their symptoms, illness or incapacitation.

Group Two consists of — The established one-third of patients whose symptoms are in part dependent on emotional factors. Psychic elaboration of the complaints add materially to the morbidity and disability.

Group Three consists of — The last one-third, a group with great diversification. In this group might be included migraine, asthma, hypertension, duodenal ulcer, nonspecific ulcerative colitis and Graves' disease. Organic disease is present. Psychic disturbances are a significant etiologic factor in the production of vegetative nervous system diseases.

The management of that vast group in which no organic pathology is found is exceedingly difficult. We can with relative ease dismiss the patient's complaints by stating that no disease is present; but this method does not eliminate the symptoms or the unserviceability and is fraught with potential danger. These patients may eventually find someone who

will treat this functional ailment by quasisomatic therapy. The concomitant pitfalls are apparent. We can achieve better results with this group by referring them to psychotherapy, but not as a punishment for our therapeutic failure. Kindness, teaching and patience will introduce them to psychotherapy where other methods fail. Unfortunately there is a great resistance on the part of many persons to admit the need for psychotherapy. This stigma must be eradicated by re-education if we are to achieve effective therapy.

The second group, in which both organic and psychic factors present themselves, most frequently confronts the otolaryngologist.

In our routine office experience we are often faced with the disparity between the apparent severity of the symptoms and the organic findings. We have all noted the severe depression and melancohlia associated with suppurative disease of the sphenoids and antra. We, too, have seen the transition to relative normal emotional behavior when the congested nose and the infection have subsided. Personality variations with severe emotional instability have been noted as a concomitant to accessory sinus disease, especially with involvement of the frontal sinuses. All intermediate stages from mild emotional crises to uncontrolled neuroses and even psychoses have been reported.

In many cases, somatic therapy alone is disappointing. Particularly is it so when incapacitating symptoms persist though the infection subsides. Psychotherapy as an adjunct to somatic therapy is the ideal approach.

The problem of tinnitus is a universal one. A patient frequently presents himself with only tinnitus as the disturbing symptom. Just as the character and the intensity of the noise may vary, so may the distress — from mild annoyance to manic or even suicidal proportion. Routine investigation seeks the etiology in hypertension, cardiovascular disturbances, endocrine disturbances, and in local pathology in the ears, nose or nasopharynx. Occasionally the pathology is

recognized, removed and the patient cured. More often, despite persistent local therapy, no alteration of the character or severity of the tinnitus occurs. The frequently associated hearing impairment adds to the emotional burden.

The psychic component of tinnitus is considerable. The distress of tinnitus may be due to these psychic equivalents. Tinnitus has a strange mystic quality — persistent, and strangely foreboding to the patient. To many it connotes a brain tumor, eventual deafness or an impending stroke. This is indeed a triple threat to the emotional balance of a sensitive human being. Constant reassurance and explanation may not reduce the intensity of the tinnitus, but may teach the patient to neglect its annoyance and to live without thought of impending calamity. Where this approach is ineffectual, psychotherapy may often be essential to prolong serviceability.

CASE REPORT.

A patient, G. R., age 48, with a menopausal syndrome, presented herself with tinnitus. A mild associated hypertension was the only finding of significance. Inquiry revealed an anxious, unstable personality whose distress from tinnitus varied with fatigue and emotional upheavals. An interesting subjective note was that she felt her emotional upheavals were precipitated by an increase in the intensity of the tinnitus.

The cycle of tinnitus and emotion kept repeating with increasing intensity and frequency until the two factors were indistinguishable. A physician-nephew denied that psychotherapy rather than tubal inflation was the conservative therapy of choice. My suggestion was met with personal hurt and anger at the insinuation that a psychoneurotic lurked in the family closet. I am told that tubal inflation was performed on the very day that the patient was found a suicide in the Hudson River.

Education which teaches that neurosis is no greater shame than mastoiditis must be instituted. Consultation between otologists and psychotherapists might have preserved a serviceable human being.

Cosmetic surgery offers another illustration. The complete personality alteration with new happiness and accomplishment has been gratifying to us following successful nasal reconstruction. None can deny the joy of the rehabilitated personality when social success accompanies a fine surgical result, but often after an apparently finely performed operation the patient returns with cosmetic complaints. Many who are at

first delighted with the result now insinuate that the surgery was unsuccessful. For them it was, because the anticipated social rebirth was never realized. Such failures can be obviated by judicious inquiry into the emotional history of the patient. Preoperatively, it is imperative for us to recognize the personalities so steeped in insecurity that even an excellent surgical result will not eliminate factors of maladjustment. Such patients are not in complete harmony with their existing status. Ofter such a patient will attribute to her misshapen nose her physical frustrations, her inability to marry and her relatively poor social or professional position. Having chosen to blame their misfortunes on a cosmetic defect, they plan and save for a plastic repair, sometimes investing their entire faith in the outcome.

To possess a reason for failure is comforting; to be deprived of it is disastrous.

Thus when the nasal plastic does not bring social success, and personality satisfaction, the patient is left with two alternatives. She must either begin to face her psychological inadequacy or persist in the belief that her nasal defect is still responsible for her continued failure. The choice she will inevitably make is obvious. To readily admit the need for psychotherapy, as we have previously noted, is painful and self-stigmatizing; therefore do we encounter the frequent demands for revised cosmetic surgery.

CASE REPORT.

I. M., aged 30, the eldest daughter of a motherless home, enthusiastically submitted to a nasal plastic. I was consulted seven months later by the patient, her father and brother. The patient felt that the previous operation was not successful; and for her it was not, for it did not fulfill her social hopes, though to an objective observer the nose was straight and cosmetically pleasant.

Suggested psychotherapy might not have been so readily rejected had not a second surgeon promised relief by revised surgery. I have traced this story through three successive operations, to culminate in a manic phase controlled by strait jacket in the office of the fourth.

Elective surgery is denied those who are considered poor surgical risks. Cardiac insufficiency and kidney dysfunction would deter us from elective cosmetic surgery. Even in non-

selective surgery we await their optimum condition before operation. So must the emotionally unstable, the frustrated, anxious neurotic personality be included as a psychically poor surgical risk. Better orientation and integration must be awaited and established, preoperatively. Cosmetic surgery performed without recognition of emotional imbalance is fraught with danger and injustice to the patient, surgeon, and to an operation which is so very gratifying when well indicated.

Another typical condition seen by otolaryngologists is that of hysterical aphonia. Since there is no ordinary organic lesion to account for this laryngeal picture of bilateral adductor paralysis, it is diagnosed as an hysterical or functional ailment. When this is determined, is it our responsibility to restore speech by any of the obvious methods of sudden fright or removal of a foreign body under general anesthesia, or is it more significant that we recognize this aphonia as just a local manifestation of a diffuse emotional disease? A symptomatic cure of hysterical aphonia without an adequate study of underlying causes, may do the patient a greater injustice than if left uncured. Such cure of an hysterical aphonia serves to drive into the deeper recesses the etiological emotional factors responsible for the somatic representation. That this hysterical symptom arises in response to an inner conflict or that this projection into illness is a flight from reality must be understood if a real cure is to be obtained by combined therapy. Later manifestations of the same emotional disease may not be so easily recognized when once an apparent cure of hysterical aphonia is affected.

CASE REPORT.

A young woman of 26, married, while her husband was in the army in the midwest became involved in an emotional affair with another man in New York. When it became necessary for her to visit her husband to make final arrangements to leave New York, she developed an hysterical aphonia. This simple infantile hysteria only necessitated cancellation of plans to join her husband. The hysterical aphonia was unfortunately cleared by the fright phenomenon. The laryngologist who treated her would have been quite content had she not developed episodes of fainting and a few days later persistent tubular vision, which, of course, is

accepted as a functional condition. Only after the psychological involvement was recognized was psychotherapy instituted. This was eventually responsible for the return of this girl to a perfectly normal existence. She is once more serviceable to her child, home and community.

Time permits me to mention but not to elaborate on many other otolaryngological conditions with psychogenic bases, or neurotic superstructures. We need but mention the well investigated and established asthma-neurotic alliance. Types of vertigo frequently identified as Ménière's or psuedo-Ménière's disease may respond well to both psychic and somatic therapy. The emotional responses of the laryngectomized patient can be traced from abysmal despair to reconciliation and eventual euphoria, under the kindly guidance and support of the psychotherapist. The indefinable and incapacitating headache, whose origin in organic disease is not obvious, may well respond to combined therapy. Such treatment should be employed long before somatic therapy alone has been determined as ineffective.

The psychological elaborations of the deafened and the deaf are great. Detailed study is now in preparation. I need but recall the irritable, irascible and suspicious personality of the deafened. The self-cloistering withdrawal from social activities is intimately tied up with these aberrations.

There are many other conditions, of course, but one deserves more than superficial mention. This is vasomotor rhinitis. There are few diseases as emotionally distressing as vasomotor rhinitis. This distress would seem to be disproportionate to the manifest local change in the nose. Yet, the functioning capacity of the patient is at a minimum at such a phase.

The causes are variously itemized as sensitivity to specific allergens, reaction to pregnancy and to menopause and the injudicious use of vasoconstrictor medications. The nasogenital relationship has been noted by many observers. The common denominator of most of these conditions is sympathetic-parasympathetic imbalance. It is virtually impossible to dissociate any of these causes from either a psychogenic basis or a psychogenic implication. In such a confused and

indistinguishable cycle, one cannot determine whether the instability is the cause or result of the vasomotor change. Case reports substantiating each are available.

The correlation of history and symptoms will often reveal the approach likely to result in well-being. Combined psychic and somatic therapy gives the greatest incidence of sustained results.

CASE REPORT.

A Spanish woman, aged 30, presented a typical picture of vasomotor rhinitis of two years' duration. Allergic investigation revealed the sensitivity to dust, followed by a protracted period of desensitization. The patient spontaneously volunteered that her nose was most congested during episodes of emotional upheaval and extreme fatigue. Her symptoms dated from the time she came to this country. No other cause was established. Local therapy, including ionization and cauterization had been tried. Benadryl during desensitization was entirely ineffectual. The patient called to cancel an appointment because her husband had fallen and fractured his leg and had been removed to a hospital. Three days after his hospitalization my patient presented herself at my office in great excitement. There had been sudden relief from the nasal congestion. This relief lasted until two days after her husband returned home. The pathway led to the investigation of her home and family and revealed a bizarre emotional and physical existence with her husband. She had married him three months before the onset of her symptoms. Her husband was a confirmed and physically terrifying alcoholic. When psychotherapy was instituted for the husband, and his orientation established, the relationship between the husband and wife improved. There was a complete cessation of all symptoms referable to the nose. This has persisted to date. My only comment, and not a facetious one, is that this woman was emotionally allergic to her husband's personality.

I must temper my enthusiasm for the psychosomatic approach with a warning. Occasionally in the absence of obvious organic disease the patient is arbitrarily classified as functional, with the sad implication of "don't take too seriously." Referrals from the badly named mental health department are often too casually inspected. In the active run of the clinic, little time is permitted for mental carthasis, with its personality revelations. Hurried investigation has its dangerous posibilities. We must not overlook organic pathology because the patient has been identified with a neurotic constitution.

CASE REPORT.

R. S., born in 1930. Had a mastoidectomy in 1936, following which the ear discharged sporadically, after acute respiratory infections. In June,

1943, the child became nauseated and vomited one morning. The doctor felt that this was an excuse for not going to school. Physical punishment thereafter followed each bout of vomiting. The child was investigated psychometrically and was discharged from one of the leading institutions as a behavior problem. There the psychologist noted "that he was of average ability but that emotional factors interfered with his efficiency. The Rorschach pattern showed an extremely constricted boy whose mode of adjustment was introversive."

The persistent vomiting in the presence of chronic otitis media with vague neurological findings indicated further study. When ventriculography was attempted a large parieto-occipital lobe abscess was evacuated.

The exquisite balance between the psyche and the soma must be appreciated and maintained. Our enthusiasm for one must not compromise the understanding of the other.

May I point out that I do not mean to take a position with any school of interpretative analysis or psychotherapy. I mean only to emphasize that the psychotherapist should be utilized by us as we would a cardiologist, dermatologist or any specialist in medicine for diagnostic and therapeutic aid. Our recognition that a patient needs and deserves such an investigation may be the greatest step in his ultimate recovery. Psychotherapy should not be resorted to only when all other approaches have failed. Rather must it be employed in conjunction with well established somatic therapy. We must be re-educated to identify pathology which might have both structural and functional complications. Coupled with our diagnostic acumen, we must have a real interest in human beings as well as in human disease. The physician and surgeon who is qualified to understand and treat psychosomatic illnesses must have as great an interest in psychobiology and psychopathology as he has in tissue pathology. We must seek and find the psychic component in organic disease. This component may be a causative factor in the production of the disease or may be an elaboration of the already established structural disease. There can be no dissociation of the psyche and the soma if the individual is to be returned successfully to health and normal serviceability.

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THE USE OF SODIUM PENTOTHAL ANESTHESIA IN ADULT TONSILLECTOMIES.*

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In many parts of the country adult tonsillectomies are performed under local anesthesia, while in others some form of general anesthesia is used. In Syracuse, general anesthesia has long held sway, apparently only by weight of popular opinion, and it is with difficulty that a physician can persuade a patient to have his tonsils removed under local anesthesia. Each patient contemplating the procedure is almost certain to say, "I prefer to be asleep during the operation," or "I don't want to know what is going on."

In the early days of my practice, an adult tonsillectomy was often an arduous and time-consuming procedure and, occasionally, the better part of an hour was required for the induction of the anesthetic and the surgery. Invariably, the induction time for the anesthesia was at least 15 minutes. Ethyl chloride or nitrous oxide was used for the induction and followed by ether. Relaxation was always a questionable entity and occasionally "strong arm" methods were necessary to keep a husky and muscular male patient on the table.

At the close of the last war, younger members of our department of anesthesia returned to private practice with a knowledge and skill in handling intravenous pentothal for anesthesia. Most of these men had had wide experience with this anesthetic agent overseas and were quick to urge its use for a multitude of minor surgical procedures.

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I discussed the possibilities of using sodium pentothal for adult tonsillectomy with them and found that at first the anesthetists were reluctant to advocate it, on the basis that laryngospasm with its undesirable sequelae was often produced. Each one agreed, however, that if blood and secretions could be kept out of the hypopharynx and larynx, that this anesthetic agent might be used successfully. Shortly after this conversation, five healthy football players were sent to us for tonsillectomies by the athletic department of Syracuse University. All five were done the same morning under pentothal with no ill effects and without difficulty. The total time for induction of anesthesia, and for the surgery for these five patients was slightly under two hours. This marked the beginning of an era in the medical circles of our city. The anesthetists and I were discussed, praised and condemned. The use of sodium pentothal for anesthesia in adult tonsillectomy is not new. A number of articles have appeared in both the British and American literature since 1942. Up to the present time, I would estimate that over 1,000 cases have been reported by various authors in the literature.

In attempting to discuss sodium pentothal as an anesthetic agent, I am not unmindful of my own ignorance concerning this drug and its actions. The details pertaining to the administration of pentothal I learned from Dr. E. Joseph Delmonico, the chief of the department of anesthesiology of the Syracuse Medical Center, who gave generously of his time and knowledge in the preparation of this paper. During the past two years I have done 183 consecutive adult tonsillectomies under sodium pentothal. The anesthesias have been carried out by the five anesthetists in the department of anesthesiology. The patient ages varied from 13, the youngest, to 76 years, the oldest. Laryngospasm did not occur. Relaxation was complete and effective. The time of induction of the anesthesia was shorter than any other type; in general, two to three minutes was the average time of induction. There were no ill effects noted in any of the cases. The indications and contraindications for sodium pentothal are much the same as for other surgical procedures with this anesthetic. Moderately severe

hypertension does not contraindicate its use inasmuch as the pressure drops somewhat under the anesthesia. As for the technique of administration, our anesthetists use a 2.5 per cent solution, or between 200 and 300 mg. Curare has been used occasionally to supplement the pentothal and to give additional relaxation of the head and neck, particularly in robust male patients. For this purpose 20 to 40 units of curare are used. It has not been found necessary to spray the throat with cocaine, pontocaine or any other anesthetic, nor has it been found necessary to infiltrate the tonsil area with procaine; however, the preoperative use of morphine and atropine, in appropriate dosage, it an important factor in maintaining a smooth anesthesia.

Dr. Ralph T. Knight, of the department of anesthesiology of the University of Minnesota, recently said, "I have given a rather large number of anesthetics for tonsillectomy with sodium pentothal, curare and nitrous oxide and I believe it to be the very best form of anesthesia for the purpose. All patients are intubated as soon as induced. We mix the pentothal with d-tubo-curarine so that each cubic centimeter of solution contains 25 mg. of pentothal and five units (0.75 mg.) of tubo-curarine. Even the little children are given this anesthetic. As soon as unconscious, they are given N_2O 65 per cent and O 35 per cent by mask, plus enough pentothal-curare until sufficiently relaxed to be intubated. From then on, they received 500 cc. each of N_2O O_2 per minute and just enough pentothal-curare to permit the surgery to be done. They are awake almost as soon as the surgery is completed. All patients are given a suitable dose of morphine and scopolamine one hour before surgery. This cuts down the dose of pentothal-curare and makes for early awakening."

Pentothal has not been used in children under 12 to 13 years of age since they do not tolerate the drug well. The most important single factor in the success of this anesthetic agent, in my opinion, is the position of the patient on the operating table. It is my custom to use the Rose position with the head lowered about 30 degrees from the horizontal. Blood

and secretions then gravitate to the nasopharynx and with the use of the Davis-Crowe type of mouth gag, the hypopharynx can be constantly observed and the airway kept free of blood and mucus. To do a tonsillectomy on an adult under pentothal in the horizontal or sitting positions is an invitation to disaster — a fact which was amply demonstrated by several of my colleagues. It is also important that the surgeon and anesthetist avoid cyanosis. For this reason, a flow of oxygen into the patient's mouth, along the blade of the mouth gag, is used. These observations of mine coincide closely with those of other men who have used pentothal for tonsillectomy and written upon this subject.

In the event that there is overdosage of sodium pentothal, sodium succinate is kept at hand to be given intravenously. In my experience this has never been necessary. In comparison to other general anesthetics, intravenous sodium pentothal possesses certain advantages for tonsillectomy:

1. The ease and simplicity of its administration.
2. The complete relaxation obtained even in large and muscular male patients.
3. The avoidance of any sense of suffocation by the patient.
4. The speed of induction of anesthesia.
5. The avoidance of postoperative nausea and vomiting.
6. The long period of postoperative somnolence which practically obviates the need for postoperative sedation.

There is one great disadvantage in the use of pentothal for tonsillectomy. This is bleeding at the time of operation. Unquestionably, bleeding is more brisk and profuse than with any other type of anesthesia. It has not been my practice to infiltrate the tonsil area with procaine either to supplement the anesthesia, which is unnecessary, or to diminish the bleeding, which leads to a sense of false security. It becomes necessary then to control the bleeding promptly and adequately by ligature or suture, or some other means. It is important that the throat be completely dry when the surgery is finished; however, this meticulous care to control bleeding at the time

of operation has apparently more than repaid the effort, inasmuch as in this series of 183 cases there were no cases of postoperative bleeding.

A second disadvantage, or rather a pitfall, which can be avoided, is laryngospasm postoperatively. In half a dozen or so of the early cases in the series, laryngospasm occurred after the patient had been transported back to his room in the hospital. There was marked cyanosis in each case. Invariably, there was some blood or mucus present in the throat and the patient was lying flat on his back. Aspiration of the secretions and oxygen was necessary to prevent disaster. In later cases, however, careful control of bleeding at operation, elevation of the foot of the bed until the patient regains his reflexes, a rubber airway to prevent swallowing of the tongue, and alertness on the part of the nursing personnel have made this complication almost nonexistent.

Conclusion: One hundred eighty-three cases of adult tonsillectomies have been reported under intravenous sodium pentothal anesthesia. This drug has several advantages over other types of general anesthesia because of ease of administration, speed of induction and freedom from distressing postoperative symptoms. Its disadvantage lies in the increased amount of bleeding that occurs at the time of operation. For its successful use in tonsillectomies, several cardinal principles must be observed; namely, the head-low position with a visible airway, a well trained anesthetist skilled in its use, supplementary oxygen to prevent hypoxia, careful hemostasis and careful bedside nursing postoperatively. In this series of cases there were no ill effects, laryngospasm did not occur at any time during operation, but did occur postoperatively in several cases. The convalescence of these patients was not altered and there were no pulmonary complications.

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THE PROBLEMS OF TONSILLECTOMY AND TRACHEOTOMY IN RELATION TO POLIOMYELITIS.*

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The problem of tonsillectomy deals with the question of whether or not this operation — which is usually coupled with the removal of the adenoids—constitutes a real danger to the patient in the incubation period or carrier state of poliomyelitis. The evidence that there is such a danger was presented in dramatic form in the "K" family of Akron.[1,2] There were six children in this family, and five of these were tonsillectomized on the twenty-second day of August. By Sept. 7 all five of these children had bulbar poliomyelitis, and three of them died. The one who was not operated upon showed no sign of illness, but the virus of poliomyelitis was recovered from the stool. There were only two scattered cases of polio in Akron prior to this family epidemic, and there were only six additional cases in this city during that September. The most probable exposure of this family was four weeks previous to the tonsillectomies during a visit with relatives in a distant community where poliomyelitis was present. Other cases have been presented where bulbar poliomyelitis followed closely upon tonsillectomy.[3]

The publication of these cases resulted in a ban on tonsillectomies in most hospitals over the country during the season of the year when poliomyelitis is prevalent; namely, the summer months. I was one of those who, as a consultant in infec-

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tious diseases, urged this ruling on the grounds that this operation is elective and could be postponed to the colder months when there was less likelihood of there being poliomyelitis virus in the nasopharynx. Pedersen[4] and others have given us some interesting food for thought on this matter. Last year I revised my opinion on this, recommending that the ideal time for tonsillectomies was immediately after the close of school in June in this part of the country; also that tonsillectomies could continue through the summer months providing there were no cases of poliomyelitis being reported from the community and no known possibility of exposure from outside. In Massachusetts last summer the State Board of Health through weekly bulletins kept hospitals informed of the situation of poliomyelitis in this state, as well as over the country at large. It would seem that this method offers a satisfactory solution to the tonsillectomy problem; nevertheless, there is always the warning of the "K" family, and regardless of season, you who take the responsibility of performing an elective operation should be aware of the fact that possible exposure at other seasons may occur. In this discussion we have come to realize that other dangers besides poliomyelitis confront this operation, and that to be forced to carry it out when upper respiratory infections are most prevalent is a disadvantage to be seriously considered. That we were carried too far in our zeal to prevent poliomyelitis through a seasonal ban on tonsillectomies is now clear, but this is just one of many errors that have been made in the numerous efforts in fighting this disease. Until we have some quick and practical method of determining the presence of the virus of poliomyelitis in patients on whom tonsillectomy is contemplated, we must rely on epidemiological data as supplied in Massachusetts.

The many errors committed in the field of poliomyelitis are attributable to the fear and panic aroused by this disease. An extreme example of this occurred in a town west of Boston. During an epidemic of polio the town fathers held conclave and decided that the best way to protect their community was to forbid the transportation of any case through this town-

ship. An ambulance hurrying a case of respiratory failure to a Boston hospital was stopped on the outskirts by a constable who forced the driver to turn back and make a wide detour. One could cite a number of equally drastic and ineffective methods that have been employed. The latest notion is the attempt to control this disease by the destruction of flies through DDT spraying, an experiment which has had no influence on the course of two well studied epidemics. You must understand that our concern regarding tonsillectomies is still very real and logical in view of the accumulated evidence which Dr. Aycock has so ably presented.

On the question of tracheotomy, my opinion remains unshaken. I have had occasion to perform many tracheotomies in the course of 30 years of service on the staff of a contagious disease hospital, and I have had the after-care of many tracheotomies performed by our surgical staff. During these 30 years I have yet to see the case of poliomyelitis that I thought required tracheotomy. This operation has been discussed by the staff on several occasions, both in connection with living cases and at the autopsy table, but never have I been convinced that the indications were present. To my mind, the indications consist of structural obstruction to the airway. In bulbar poliomyelitis I have not been confronted with structural obstruction either from swelling of the parts or spasmodic contractions. The obstruction which occurs is from secretions. The patient cannot swallow, cannot close the glottis, and is, therefore, unable to cough. Postural drainage and gentle, skillful suction are indicated. Atropine, in my experience, does not stop these secretions; it merely dries it into a consistency which makes it more difficult to get rid of. I can imagine a spasm of the vocal cords which could give rise to a crowing inspiration, but I have never encountered this.

At the First International Poliomyelitis Conference in New York last July the subject of tracheotomy was defended by Baker[5] and Priest in the same manner that the subject was presented at the Rocky Mountain Conference in Denver in 1946. In the discussion which followed, there was criticism

of the indications given and especially of the readiness to perform the operation early to prevent future obstruction through laryngeal stridor. It was pointed out that these authors failed to emphasize the value of postural drainage. Wilson, who had been a pioneer in advocating this operation when he was at the Boston Children's Hospital, felt that the operation had been effective in saving the life of a very few patients, but he could not accept the indications for this operation as expressed by Priest and Baker. In the opinion of Wilson, the chief indication consisted of extreme restlessness due to inability to establish sufficient airway by means of postural drainage and suction. Stimson, Shaw, Pierce and Anderson, all with extensive clinical experience in all phases of this disease, expressed a reluctance to endorse the enthusiasm for tracheotomy emanating from the Minneapolis group. It is impossible to gauge the value of this operation when performed early, because there is no way of knowing whether it actually saved a life or whether the patient recovered in spite of the added risk of the operation. Tracheotomy is a hazardous undertaking. Perhaps those of us who have had no experience with tracheotomy in bulbar poliomyelitis are not in a proper position to speak against it. It is always possible that the Minneapolis cases presented a peculiar genius epidemicus that warranted this operation, but this is certainly not made clear to most of us who are familiar with the indications and value of tracheotomy in other infections and with the care of the many phases and manifestations of bulbar poliomyelitis.

It is appropriate at this time to recall the waves of undue enthusiasm for certain measures which have led us astray in our efforts to deal with poliomyelitis: convalescent serum therapy with intrathecal injections; drastic immobilization of the extremities with casts and splints; the Kenny concept; prostigmine, heralded by de Kruif's article in the *Reader's Digest*, "They Shall WalkAgain"; and Ransohoff's curare and muscle stretching. We have been through all of these phases and have had to rearrange and modify our ideas through

extensive experience. In view of this, it is not surprising that many of us look with misgiving on what today appears to be an over-emphasis of tracheotomy in bulbar poliomyelitis.

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CANCER OF THE FACE; TREATMENT AND PLASTIC REPAIR.*

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Patients with cancer of the face do not receive as efficient treatment as one would expect from the site of the lesions and the facility with which a diagnosis can be made. When a diagnosis can be definitely established by microscopic examination of either a fresh frozen section or a fixed section, it is difficult to understand why so many lesions about the face are treated in a conservative way. In cases in which this type of treatment is employed, the growth frequently recurs and becomes fixed to cartilage or bone, or metastasizes to the neck. These complications make the necessary treatment much more radical, produce postoperative defects and make the outlook for controlling the growth very questionable. Although biopsy should be performed before treatment is instituted in cases in which the diagnosis is questionable, it is much better to remove the entire growth, if possible, and to examine a fresh frozen section to corroborate the clinical diagnosis. I do not think that a specimen for biopsy should be removed by a physician who is not prepared to treat the tumor in a radical way if it proves to be malignant. The chance for permanent cure will be much better if the surgeon who is to do the operation can examine the patient before a specimen for biopsy is removed so that he is able to determine the apparent extent of the tumor and also to select the site from which the specimen should be removed. At times, the trauma resulting from

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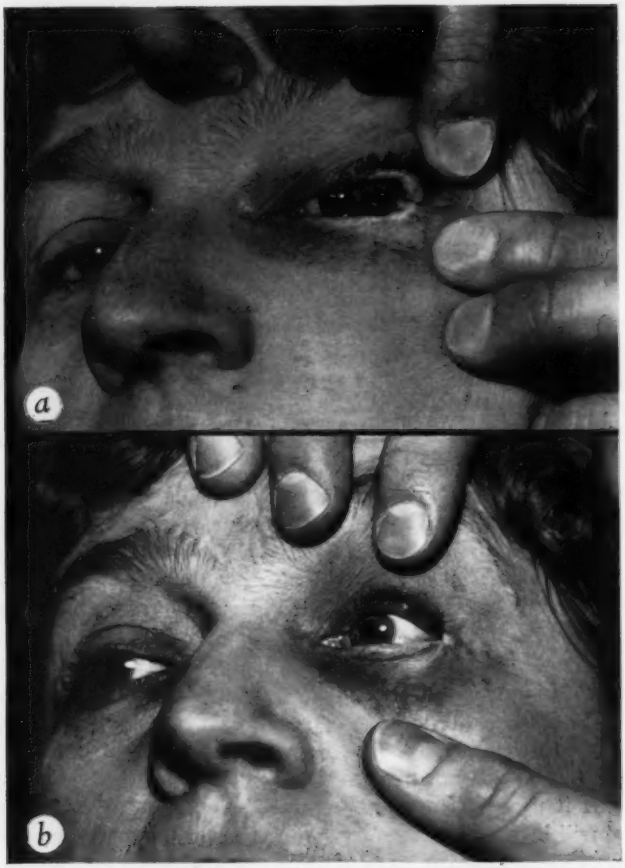

Fig. 1. (a) Postirradiated basal-cell epithelioma of the outer third of the upper and lower eyelids, and the outer canthus fixed to the orbital conjunctiva. The lesion was removed by surgical diathermy. (b) Postoperative appearance five months later. There was no recurrence after six years.

the removal of the specimen makes it impossible to determine how much of the enlargement is the result of the tumor and how much is the result of reaction from the trauma caused by removal of the specimen. This makes it difficult for the surgeon to decide how widely to remove the tumor beyond the

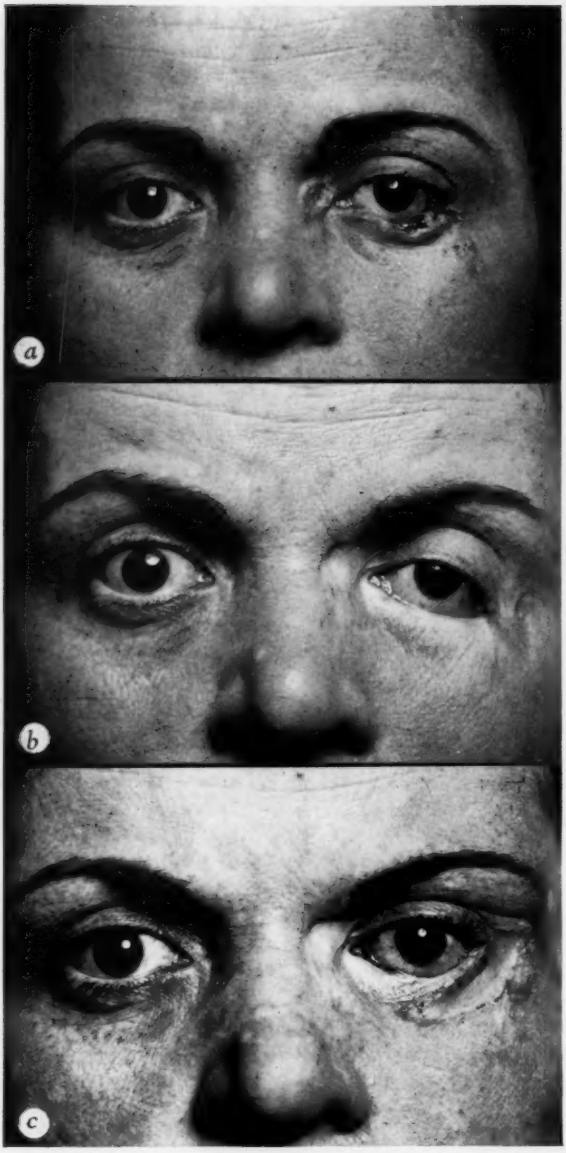

Fig. 2. (a) Postirradiated basal-cell epithelioma of the entire lower eyelid, outer third of the left upper eyelid and inner canthus, with an area of involvement below the eyelid on the left cheek. The lesion was removed by surgical diathermy. (b) Postoperative appearance 11 months later. (c) Appearance 19 months later. An advancement flap was made from the left cheek, and then a skin graft was used to correct the deformity.

apparent margin. Sometimes, in removing the specimen for biopsy, the incision is placed so that it interferes with a pedicle flap or a rotation flap that might be employed in a reparative procedure. In other words, if a plastic operation

Fig. 3. (a) Postirradiated basal-cell epithelioma of the orbit and forehead. It was necessary to remove the eye and the growth by surgical diathermy. The dura was exposed over an area 2.5 cm. in diameter. (b) Postoperative sequestrum. (c) Wound preliminary to grafting. (d) Subaxillary abdominal flap was elevated in two portions. (e) The two tube flaps were joined together and brought up in stages to cover the postoperative defect. (f) Postoperative appearance two years after removal of the growth.

ultimately will be required, the plastic surgeon should be consulted regarding the tumor before its removal, or, better still, he should be allowed to remove the lesion. Cancer about the skin of the face may be a basal-cell epithelioma, or both a basal-cell and a squamous-cell carcinoma. Although the grade of malignancy of a squamous-cell epithelioma usually is low, it occasionally may be high. At times, one sees a squamous-

cell epithelioma that simulates a sarcoma and grows very rapidly. This type of tumor usually occurs in actinodermatitis which follows irradiation. While the microscopic appearance indicates that this type of tumor is very malignant, I have never seen a case in which the patient did not stay well after wide removal of the tumor with surgical diathermy. This tumor rarely metastasizes. Adenocarcinomas of the mixed tumor type or of the cylindroma type may occur in the parotid region.

Fig. 4. (a) Actinodermatitis (precancerous) of the nose. The affected tissue was removed and a forehead flap was brought down to replace the defect. (b) Postoperative appearance.

In the surgical management of malignant tumors of the face, either sharp dissection or electrocoagulation (surgical diathermy) may be employed. When using these two methods of treatment, cancer of the face may be divided into four groups: *1.* lesions which permit of sharp excision and which, after excision, leave wounds that can be closed immediately by simple approximation of their edges; *2.* lesions which permit of sharp excision and which, after excision, leave wounds that require the use of a skin graft for repair; *3.* lesions which should be removed by surgical diathermy and which,

after electrocoagulation, leave small wounds that heal spontaneously by granulation and epithelialization (see Fig. 1); and 4. lesions which should be removed by surgical diathermy and which, after electrocoagulation, leave extensive facial defects that require some form of plastic repair (see Fig. 2). Figs. 1 through 11 illustrate the treatment and plastic repair of cancer of the face.

Fig. 5. (a) Actinodermatitis (precancerous) of the right cheek and neck. The affected tissue was excised and replaced with a full-thickness skin graft. (b) Postoperative appearance. Partial excision and advancement flaps would reduce the size of the graft.

The first group includes small basal-cell and low-grade squamous-cell epitheliomas of the face which have not been treated previously and which are so situated that primary closure of the wound is possible after wide removal of the growth (see Figs. 8 and 9). For instance, many small lesions situated about the forehead, eyelids, lips and cheeks can be treated in this manner. Also included in this group of tumors are freely movable adenocarcinomas of mixed tumor type in the parotid region.

The second group of tumors includes basal-cell and squamous-cell epitheliomas which are large, which have not been

treated previously, and which are superficial and freely movable; after removal, however, the resultant wound is too large to permit appproximation of the skin edges and, consequently, must be covered with a skin graft (see Figs. 5 and 6). Either

Fig. 6. (a) Bilateral actinodermatitis of both cheeks, upper lip and chin, with multiple areas of epithelioma which were removed and replaced with skin grafts. While a delayed or tubed flap might have been employed in this case, the character of the skin and the subcutaneous tissue would not permit this. (b) Postoperative appearance.

a free full-thickness or shaved skin graft is employed for this purpose; usually, I prefer a full-thickness dissected graft. If the denuded surface is not too large, a full-thickness skin graft obtained from behind the ear or from the mastoid region is preferable, because its color and textures are very nearly the same as those of the skin of the face. In larger areas, a full-thickness skin graft from the supraclavicular region is to be recommended. In some instances, a more satisfactory repair can be accomplished by means of a small pedicle flap of skin which is advanced over the defect from the adjacent tissues (see Figs. 4 and 11).

The third group of tumors includes small malignant lesions which are of a high grade of malignancy or which recur after previous treatment, particularly irradiation therapy.

Fig. 7. (a) Postirradiated basal-cell epithelioma of the upper lip fixed to the upper jaw and nose. This was removed by surgical diathermy. (b) Appearance 14 months later. (c) One year later a lined sickle flap was employed to reconstruct the upper lip.

The fourth group of tumors includes very extensive malignant lesions of the face which have been treated previously. The most serious members of this group are those skin cancers

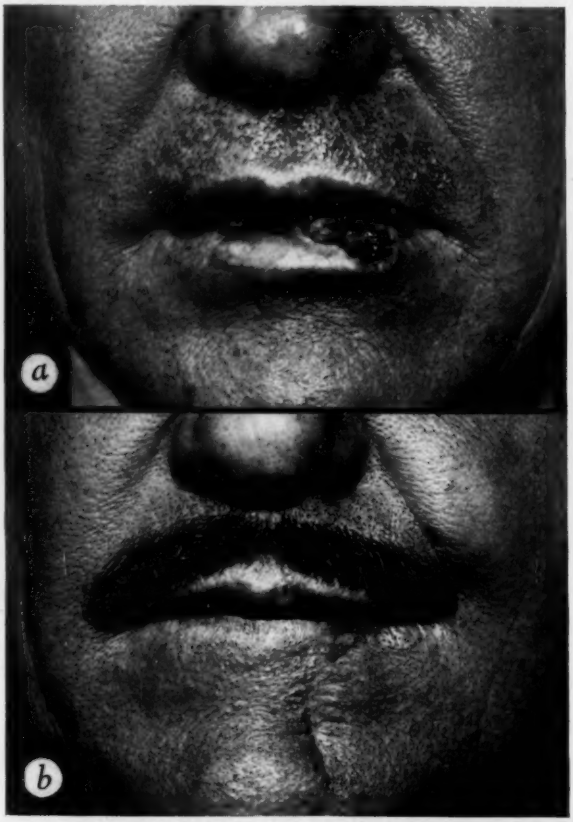

Fig. 8. (a) Postirradiated squamous-cell epithelioma, Grade 2, of half of the left lower lip. (b) The epithelioma was excised and a plastic operation was done at the left angle of the mouth. This was followed by a dissection of the submental and submaxillary glands.

which recur after previous irradiation therapy and which are fixed to underlying cartilage or bone about the orbits, jaws, cheeks, ears or nose (see Fig. 7). Under these circumstances,

the growth has infiltrated the soft tissues down to the periosteum or perichondrium and may actually be invading the bone or cartilage. Postoperative fixed adenocarcinoma of the parotid region is also in this group. I believe that the treatment of choice in such cases is wide removal with surgical diathermy and thorough electrocoagulation of the involved perichondrium or periosteum far beyond the apparent limits of the growth.

Fig. 9. (a) Epithelioma of the entire lower lip. The patient had had an epithelioma removed from the right cheek and lower lip several years previously. A bilateral dissection of the submental and submaxillary glands was done here. (b) Postoperative appearance. The growth was excised and a plastic operation was done at either angle of the mouth and upper lip. The stitches have just been removed.

In cases in which a carcinoma recurs after irradiation and involves cartilage or bone, the tumor may be found by microscopic examination of fresh frozen sections of tissue far beyond the apparent limits of the growth; therefore, if tissue about the growth appears to be involved, specimens should be removed and examined in the course of the operation.

This method of having an examination made, during operation, of fresh frozen sections of questionable tissue about the growth, has been used at the clinic for many years and has

given the most satisfactory results in cases in which recurring malignant growths involve bone or cartilage. In these cases, a sequestrum usually occurs and has to be removed in two months after the operation, when it can be separated from the normal bone. The removal of growths about the face

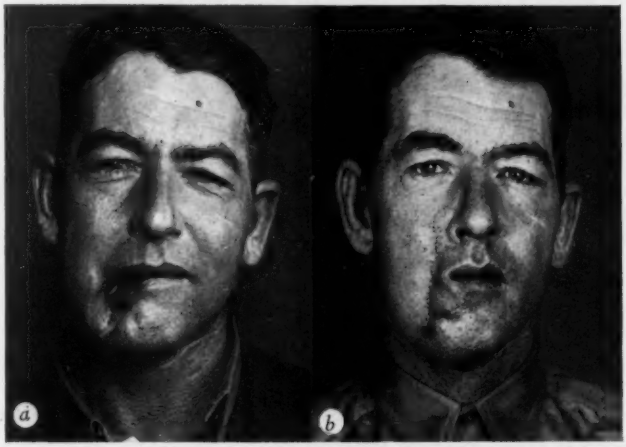

Fig. 10. (a) Postoperative and postirradiation defect associated with squamous-cell epithelioma, Grade 2, of the right lower lip and jaw. A large sequestrum that extended from the midline to the region of the last molar tooth was removed from the right side of the lower jaw. An Eslander type of operation was done to fill in the postoperative defect. (b) Postoperative appearance seven months later. The stitches have just been removed. A secondary operation was done to enlarge the angle of the mouth on the right side.

sometimes leaves large openings in the frontal sinus, ethmoid cells, maxillary sinus, nose, lips and cheek (see Fig. 10). In cases in which the tumor is active, it is best not to attempt reconstruction until at least a year from the time of the removal, in order to be fairly certain that recurrence does not take place. If a flap or a free graft is used to close a defect shortly after operation, particularly in cases in which the tumor is active or has recurred after irradiation, and if some thickening of the graft occurs, it is difficult to determine whether or not the tumor has recurred. The tumor sometimes recurs underneath the graft or flap before the patient or the surgeon is aware of it. It is advisable to make fairly sure

the patient is well before the defect is repaired. If this is done one can be reasonably certain that there will not be recurrence.

If the bone of the forehead, skull or malar region has been destroyed, it may be possible to chisel away the sequestrum and allow granulation tissue to come up through the normal

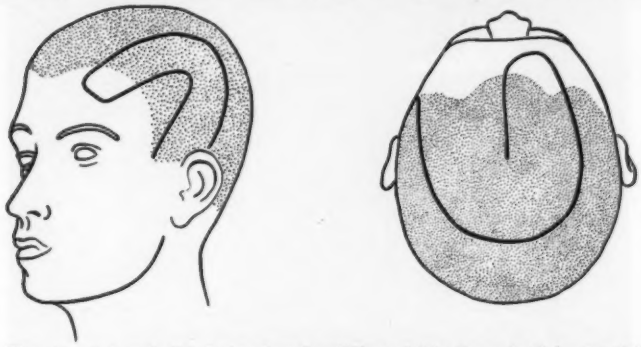

Fig. 11. (a) and (b) Lateral and midline sickle flaps used in repairing postoperative defects about the face after removal of carcinoma.

bone and then apply a split-skin graft. In cases in which the defect is large, various types of pedicle flaps can be employed. The common ones for the smaller defects are the lateral and midline sickle flaps. For the larger defects, submaxillary, supraclavicular or subaxillary abdominal flaps may be employed (see Fig. 3).

Complete primary removal of carcinoma of the face is essential. Examination of fresh frozen sections of tissue at the time of the operation aids in determining the best treatment. In cases in which the tumor recurs and becomes fixed after irradiation, examination of fresh frozen sections is an aid in determining the limits of the growth. Bilateral dissection of the submental and submaxillary glands should be done in cases of squamous-cell epithelioma, Grade 2, 3, or 4, of the lower lip or cheek. The time of repair of the postoperative defects should depend on the nature of the growth and on the previous treatment. Irradiation, if used at all, should be a secondary procedure.

EPISTAXIS.*

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INTRODUCTION.

The literature on epistaxis contains many case reports and descriptions of various preferential techniques for handling nasal hemorrhages.[1-39] There have been, however, few comprehensive studies which consider the anatomy, histology and pathology of epistaxis as it relates to the clinical problem.[40-43]

It is the aim of this study to evaluate the incidence, cause and control of nasal bleeding seen in a large hospital center where both children and adults were observed.

ANATOMY.

The most frequent bleeding site on the septum is located on the extreme anterior inferior portion of the nasal septum and is known as Little's or Kieselbach's area.† The vascular plexus of Little's area is composed of various sized arterioles, capillary and venous channels within the mucous membrane. This plexus is supplied by the nasopalatine branch of the sphenopalatine artery, the terminal branches of the descending palatine and superior labial arteries, and the branches of the anterior and posterior ethmoidal arteries (see Fig. 1).

*This work was made possible by grant from the Upjohn Co., Kalamazoo, Mich.

†Investigation by McKenzie[43] revealed that the anatomical description of this area by Little preceded that of Kieselbach.

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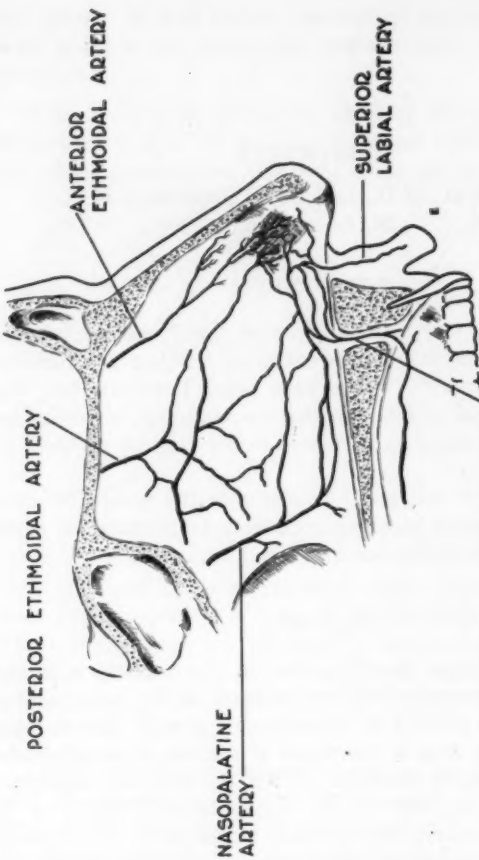
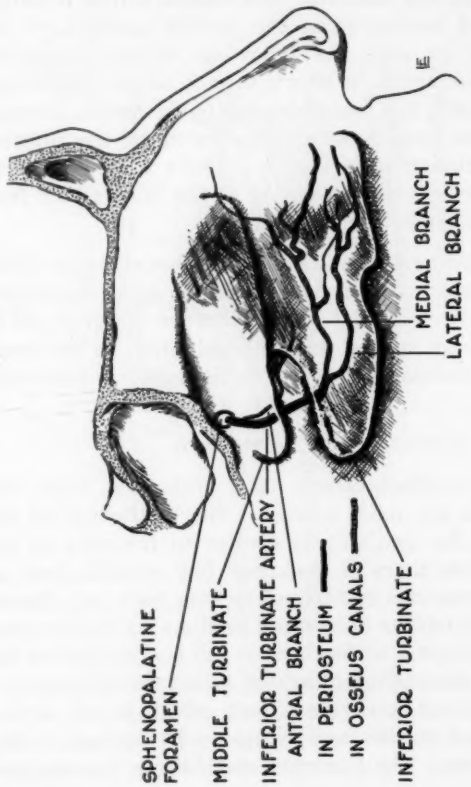


Nasal Septum Showing Arterial Supply of Little's Area
FIGURE I

Bleeding from the lateral nasal wall has received insufficient attention in the literature, so a review of the vascular supply of the lateral nasal wall is presented in some detail. The anterior and posterior ethmoidal arteries supply the anterior and superior parts of the lateral nasal wall, while the branches of the sphenopalatine artery distribute themselves to the posterior and inferior portions.

Burnham[40] has investigated and described in great detail the distribution of the branches of the sphenopalatine artery, *viz.*, *a.* the inferior turbinate, *b.* middle turbinate, and *c.* naso-palatine arteries.

a. The inferior turbinate artery courses downward and forward in the periosteal layer, giving off variable branches to the common meatuses. When it reaches the posterior tip of the inferior turbinate, three terminal vessels are given off and pass through bony canals, emerging at the central one-fifth of the inferior turbinate bone. These three canals with



LATERAL NASAL WALL SHOWING DISTRIBUTION OF INFERIOR TURBINATE ARTERY AND BRANCHES (MODIFIED FROM BURNHAM)

FIGURE II

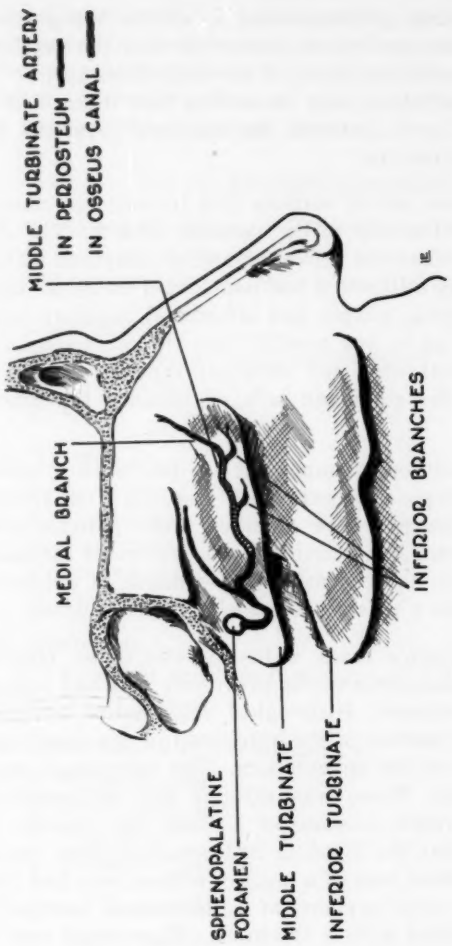
their contained vessels are as follows: *1.* The canal for the antral vessels passes along the junction of the inferior turbinate with the lateral nasal wall, and is patent along its upper nasal border; *2.* The supero-medial canal lies below the antral canal and terminates anteriorly on the medial surface of the inferior turbinate; *3* The infero-lateral canal lies immediately below and emerges on the lateral surfaces of this bone (see Fig. 2).

b. The middle turbinate artery leaves the sphenopalatine artery and reaches the under surface of the middle turbinate. At the junction of the posterior and middle thirds it enters a bony canal and breaks up at the central one-fifth of the middle turbinate to give the following constant terminal branches of this artery: *1.* a lateral branch to the lateral surface of the turbinate, *2.* a medial branch to the medial surface, and *3.* an inferior branch which courses along the inferior border of the turbinate (see Fig. 3). There is also a fourth variable branch supplying the region of the bulla which may arise near the sphenopalatine foramen.

c. The nasopalatine artery leaves the sphenopalatine foramen and courses along the attachment of the middle turbinate supplying posterior ethmoidal cells. Branches are given off to the anterior inferior face of the sphenoid sinus, to the nasal septum, to the sphenoid ostium, and to the superior turbinate.

HISTOLOGY AND PATHOLOGY.

At the nasal vestibule there is a transition from the skin of the lip to the nasal mucosa. The epithelium of the nasal portion of the vestibule is similar to the skin of the lip. Central to this there is a narrow but variable area of nonkeratinized squamous epithelium lacking hair and glands. Vascular tufts resembling flat topped papillae which are present towards the external orifice become tall and slender as the nasal mucosa is approached. Each of the terminal points of these papillae contain a capillary loop which arises in the highly vascularized corium and comes to lie against a thin basement membrane. The basement membranes become pro-



LATERAL NASAL WALL SHOWING DISTRIBUTION OF MIDDLE TURBINATE ARTERY AND BRANCHES (MODIFIED FROM BURNHAM)

FIGURE III

gressively thinner as the capillary comes into close apposition with the epithelium until they can no longer be separated individually. The capillary tuft is accompanied initially by connective tissue fibrils which are lost as the vessel approaches

the surface. Stratum germinativum is clearly recognizable above the basement membrane, indicating that the capillary loops lie below the deepest layer of the epithelium proper. In some cases the epithelium may be so thin that there may be only several cell layers between the capillary loop and the surface of the epithelium.[41]

The nasal mucosa which borders this transitional zone is in even closer relationship to the vascular system. The epithelium is pseudo-stratified columnar and is composed mainly of mucus producing cells and a few nonciliated columnar cells. In the tunica propria, simple and bifurcated capillary loops extend diagonally up to and possibly into the epithelium. At the most superficial area the terminal capillary loops lie directly between the columnar cells or beneath the mucus producing cells.[41]

Dohlman[42] considered it important to determine whether there was a pathologic explanation for bleeding from Little's area. Biopsy specimens were obtained from patients who bled habitually from Little's area and the specimens sectioned serially in order to secure pathologic evidence in epistaxis. From these sections a plastocene model was constructed.

In one of Dohlman's cases, a biopsy was taken from a patient who had bled three weeks previously and had had no previous local treatment. Histological examination revealed a thrombus on the surface of the outer wall of the vessel and over a small part of the endothelium. The remaining endothelium was intact. There was little if any inflammatory reaction. A remarkable absence of healing was present, in view of the fact that the bleeding had occurred three weeks previously. In another case of a healthy woman who had bled for several days, serial sections of Little's area revealed a torn blood vessel filled with a thrombus. This vessel was in connection with a deep thrombosed venous plexus. Round cell infiltration was present around the superficial thrombus. In several places, the epithelium which covered the capillaries seemed altered and thus offered poor protection to the capillaries.

The relative absence of inflammation, the poor healing and the presence of deep venous plexus thrombosis led Dohlman to believe that there were important "endogenous factors" in addition to exogenous factors which contribute to the initiation and recurrence of nasal bleeding.

CAUSAL FACTORS IN EPISTAXIS.

It is beyond the scope of this report to discuss the many factors involved in the etiology of epistaxis; therefore, only the more common diseases with associated epistaxis are listed.

A. Inflammatory:
1. Acute coryza (vigorous blowing).
2. Rhinitis sicca.
3. Ozena.

B. Traumatic:
1. Fracture of nose.
2. Ulceration of septum due to picking.
3. Post-operative.
4. Instrumentation of sinuses.
5. Foreign bodies.

C. Mechanical:
1. Deviated nasal septum.
2. Nasal polyps.

D. Disturbances of Circulation:
1. Hypertension.
2. Arteriosclerosis.
3. Nephritis.
4. Telangiectasis.

E. Blood Dyscrasias:
1. Polycythemia.
2. Leucemia.
3. Purpura hemorrhagica.
4. Hemophilia.
5. Splenic anemia.
6. Anemias.

F. Infectious Diseases:

1. Rheumatic fever and rheumatic heart disease.
2. Scarlet fever.
3. Whooping cough.
4. Smallpox.
5. Diphtheria, nasal.
6. Typhoid and paratyphoid fever.

G. Granulomata:

1. Tuberculosis.
2. Syphilis (Gumma of nose)
(Congenital syphilis).
3. Lupus erythematosus.

H. Neoplasm:

1. Benign.
2. Malignant.

I. Vitamin Deficiency.

1. Scurvy.
2. Vitamin K deficiency.

J. Miscellaneous:

1. Metabolic disease.
2. Liver disease.
3. Vicarious menstruation.
4. Chemical poisoning (Chrome, mercury, phosphorus).
5. Salicylism.
6. "Endogenous factors."

METHODS PROPOSED FOR THE CONTROL OF NASAL HEMORRHAGE.

Because of the disparity of methods used for the control of nasal hemorrhage, only an outline of the techniques will be presented.

I. Medical:

A. Pressure.

1. Pressure pack (cotton or vaseline gauze).
2. Traction pack (Beavis[1]).
3. Intranasal balloon (Stevens[2]), (Ford[3]).

B. Coagulants.

1. Thrombin (Tidrick, *et al.*[4]), (Stevenson[5]), (Fox[6]).
2. Fibrinogen (Effler[7]).
3. Hemoplastin (Brown[8]).
4. Coagulin (Brown[8]).
5. Claudin (Brown[8]).
6. Vitamin K.
7. Koagumin.
8. Salt pork (Cone[9]).
9. Absorbable sponge.
10. Oxycel (Houser[10]).

C. Vasospastic Agents.

1. Adrenalin.
2. Posterior pituitary extract (McLaurin[11,12]).
3. Forced inspiration (Dundas Grant[13]).
4. Ice.

D. Cautery.

1. Chromic acid.
2. Silver nitrate (Littel[14]).
3. Trichloracetic acid.
4. Electrocoagulation.

E. Sclerosing Agents.

1 Quinine lactate (Monson[15]).
2. Sylnasol (Fox[16]).
3. Ten per cent phenol in almond oil (Asherton[17]).
4. Five per cent quinine in urethane (O'Kane[18]).

F. Iontophoresis (Beck[19]).

G. Radium (Scal[20]), (Weiss[21]).

H. Unknown.

1. Ovarian hormone (Connell[22]).
2. Viper venom (Higgins, *et al.*[23]).
3. Moccasin venom (Goldman[24]), (Dack[25]).

II. Surgical:

A. Submucous resection (Rainey[26]).

B. Submucous elevation (Lyman).

C. Ligation.

1. Anterior ethmoidal artery (Goodyear[27,28]), (Windham[29]).

2. Septal branch of superior labial artery (Rosenvold[30]).

3. Internal maxillary (Hirsch[31]).

4. External carotid (Hyde[32]), (McKnight[33]), (Abrahams[34]), (Hawthorne [35]), (Bricker[36]), (Johnson[37]), (Barker[38]), (Spar-Hallberg[39]).

HOSPITAL SURVEY OF EPISTAXIS.

A survey was made of the hospital records of all cases of epistaxis admitted to Barnes Hospital and Children's Hospital between 1928 and 1942. The study comprised 88 adult cases and 48 children's cases. Only those records were utilized in which complete examinations had been made and accurate data obtained. In all cases a diagnosis of epistaxis, severe enough to require hospitalization, was established with the exception of some of the children's cases where epistaxis occurred incidental to hospitalization for rheumatic fever or rheumatic heart disease. For purposes of analysis, the cases were divided arbitrarily into the following three age groups: *1.* children's group (under 15 years); *2.* young adult group (between the ages of 15 and 40); and *3.* an older adult group (between 40 and 80 years).

It will be shown that there are important differences in the three groups with respect to site of bleeding, etiology and type of therapy demanded.

In Chart 1, it is seen that in the two adult groups the incidence of epistaxis among males was higher than among females. It is particularly striking in the older adult group. This is in contrast to the children's group where females were somewhat more affected than males. This may be explained in part by the fact that rheumatic fever and rheumatic heart

TABLE 1. LATERALIZATION OF BLEEDING IN RELATION TO AGE GROUPS.

Lateralization	Number of Cases		
	0-15 Yrs.	15-40 Yrs.	40-80 Yrs.
Left Side of Nose	11	21	27
Right Side of Nose	8	12	15
Bilateral	14	6	4
Side Not Stated	15	3	0
Total	48	42	46

TABLE 2. CLINICAL DIAGNOSES ASSOCIATED WITH EPISTAXIS.

Clinical Diagnoses	Number of Cases		
	0-15 Yrs.	15-40 Yrs.	40-80 Yrs.
Rheumatic Fever (Active)	16	—	—
Rheumatic Heart Disease (Inactive)	8	—	—
Hypertensive Cardiovascular Disease	—	7	27
Generalized Vascular Sclerosis	—	—	28
Chronic Glomerulonephritis	—	1	—
Leucemia (All Forms)	4	2	1
Aplastic Anemia	—	1	—
Telangiectasis	—	1	—
Purpura Hemorrhagica	1	1	—
Granuloma of Turbinate	—	1	1
Plasma Cell Myeloma	—	—	1
Sphenoethmoid Carcinoma	—	—	1
Syphilis (Congenital or Active)	1	1	—
Infectious Mononucleosis	—	1	—
Maxillary Sinusitis	—	1	1
Dean Needle Irrigation for Diagnosis	—	2	—

Cases in which the Clinical Diagnoses were unrelated to Epistaxis were not included in this table.

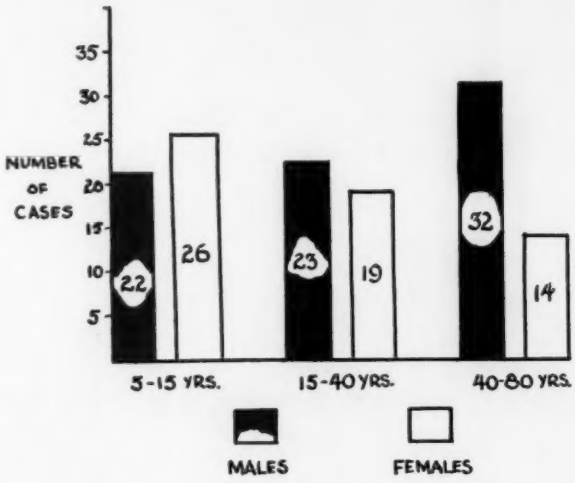
TABLE 3. LOCATION OF BLEEDING IN RELATION TO AGE GROUPS.

Bleeding Site	Number of Cases		
	0-15 Yrs.	15-40 Yrs.	40-80 Yrs.
Little's Area	48	30	20
Inferior Meatus, Anterior	—	3	2
Inferior Meatus, Posterior	—	—	6
Inferior Turbinate, Posterior	—	3	1
Middle Meatus, Posterior	—	4	5
Middle Turbinate, Posterior	—	1	1
Superior Meatus, Posterior	—	—	2
Posterior (Exact Site Not Stated)	—	1	10
Posterior Septal Spur	—	—	4
			*

*Multiple Bleeding Points explain discrepancy in totals.

CHART I

Sex Distribution of Epistaxis in Relation to Age Groups



disease, which are responsible in this study for 50 per cent of the cases of epistaxis in the children's group, affects females twice as often as males.

Chart 2 shows that in this survey the problem of severe epistaxis in a hospital is most often encountered in individuals between five and 25 years. This finding is related no doubt to the high incidence of rheumatic fever in younger individuals. The incidence then tapers off and remains uniform in the young adult group. There is some increase beyond the age of 50 and a sharp drop past the age of 75. It is interesting to note (see Table 1) that in this study bleeding occurred more often from the left side of the nose in all age groups. Although this difference is slight in the children's group, the predominance is very marked in the adult group.

CHART II

Distribution of Epistaxis According to Age

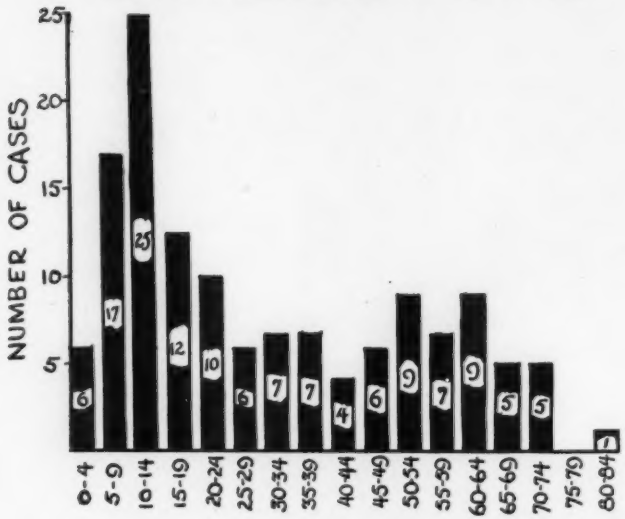


Table 2 represents a summary of all of the clinical diagnoses which were available at the time of this study which might be presumed to bear a causal relationship to severe epistaxis. Those cases in which the clinical diagnoses were unrelated to epistaxis were not included in this table.

In the children's group, active acute rheumatic fever and inactive rheumatic heart disease represented 50 per cent of the cases (24 out of 48 cases). Recurrent bleeding was twice as common in the active phase as in the inactive state of rheumatic heart disease. Leucemia was the causative factor in four cases; congenital syphilis and purpura hemorrhagica each accounted for a single case.

Among the cases in the two adult groups, the rheumatic diathesis did not account for a single case of epistaxis. In

the young adults, hypertension was noted in seven cases, while in the older adults this number was significantly increased (27 cases). As would be expected, generalized vascular sclerosis predominated in the older adult groups (28 cases). In addition a variety of associated clinical diagnoses was noted in both adult groups.

In evaluating the site of bleeding (see Table 3), it was considered desirable to classify bleeding as anterior or posterior in origin, as well as the usual designation of lateral and septal; therefore, the nasal fossae were arbitrarily bisected by a vertical plane, drawn approximately between the anterior and posterior tips of the inferior turbinates.

In the children's group, all bleeding was anterior and septal. Not a single case occurred from the lateral nasal wall or from the posterior half of the nose.

In the young adult group, approximately 80 per cent of all bleeding occurred from the anterior part of the nose. The remaining cases were posterior in origin from the region of inferior and middle turbinates and the middle meatus.

In striking contrast, bleeding in the older adult group is posterior in origin in over half of all cases. Twenty-two cases of anterior bleeding and 29 posterior sites of bleeding were observed. Anterior bleeding was almost always septal and usually arose from Little's area. With the exception of four cases of bleeding which originated from posterior septal spurs, all posterior bleeding developed from the lateral nasal wall. The inferior, middle and superior meatuses were the primary sites of this bleeding (13 cases); in 10 instances, the exact site was not stated. The inferior and middle turbinates were each involved in one case.

TREATMENT.

The following were the methods of treatment used:

1. Anterior nasal packing (cotton, cotton saturated with adrenalin, vaseline gauze, tannic acid and adrenalin, and salt pork wedges).

2. Post-nasal packing.
3. Cauterization (chromic acid, trichloracetic acid, silver nitrate and electrocautery).
4. Submucous elevation and submucous resection.
5. Ligation of external carotid.
6. Caldwell-Luc operation.

Regardless of the type of packing used, anterior nasal packing was usually effective in controlling anterior nasal bleeding. Similarly, cauterization by chemicals or electrocautery effectively sealed off the bleeding vessel when the bleeding point was localized and accessible.

Submucous elevation was performed in nine children. These cases were observed for a varying period of six months to two years. In seven cases bleeding did not return during this period of observation. In the eighth case bleeding was reduced in frequency. In one case there was inadequate follow-up.

Submucous resections were done for intractable septal spur bleeding in one adult under 40 years and one over 40 years with good results for the duration of hospitalization. In one case of bleeding from the maxillary sinus, a Caldwell-Luc was necessary for control of the hemorrhage. Ligation of the external carotid artery effectively controlled posterior bleeding in four instances after all other measures had failed.

In some cases of nasal hemorrhage, adequate control of the bleeding could not be obtained without the use of a post-nasal pack employed in conjunction with anterior packing. Post-nasal packing was not required in any of children's cases, but was necessary in the young adult group in three of the nine cases with posterior bleeding. In the older adult group, however, a combination of post-nasal and anterior packing was required in 16 of the 24 bleeders.

Otitis media is an important possible complication which may result from nasal packing. This occurred in only one case in each age group where anterior packing alone was used (three out of total 103 cases). In contrast, when post-

nasal packing was used, the incidence of otitis media was higher. In the young adult group, two of the nine cases developed otitis media. In the older adult group, two of the 16 patients developed otitis media when the posterior packing was allowed to remain in place for five days.

DISCUSSION.

In evaluating the incidence, causes, location and control of epistaxis, it is essential that cognizance be taken of certain data not included in this study. The incidence of bleeding reported here represents a hospital case study and does not necessarily take into account the mild to moderate bleeders treated at home or in the office.

Fifty per cent of recurrent epistaxis in the hospitalized children was associated with active or inactive rheumatic heart disease. Thus, in rheumatic children, bleeding may be an important otolaryngologic problem. The high incidence of epistaxis among children in this study is a result, at least in part, of the large number of rheumatic fever cases observed in St. Louis Children's Hospital. It is our impression that in private practice there is also a higher incidence of epistaxis in children than in adults. It is not to be assumed, therefore, that epistaxis occurring in children is pathognomonic of rheumatic fever; however, rheumatic fever should not be overlooked when epistaxis is associated with generalized systemic complaints.

In this hospital study we did not find an association of epistaxis and rheumatic heart disease in the young adult or older adult cases. This would appear to suggest that nasal bleeding does not occur frequently among adult rheumatics, or, if it does occur, it is easily controlled by the patient or the physician in charge of the cardiac problem, and is therefore not coded and filed as a principal diagnosis.

In the older adult group, bleeding is frequently associated with hypertension and generalized vascular sclerosis in this series. It is shown that well over 50 per cent of those individuals in this age group entering the hospital with nasal

bleeding have hypertension.* The obvious conclusion follows that in uncomplicated bleeding of older individuals hypertension is to be suspected. When bleeding occurs in this older group it is posterior in origin in more than half of the cases. In contrast, bleeding is invariably anterior in children. We are unable to account for this difference unless it is related to hypertensive (and) or degenerative changes in the complex vascular distribution of the posterior lateral nasal wall as described so well by Burnham.[40] The larger arteries of the lateral nasal wall are at least in part of their course in a relatively exposed position beneath the mucosa of the common meatuses and the middle and inferior turbinate region. It is conceivable that at such points, where the vessels are superficial and weakened by degenerative changes, that severe hemorrhage can easily occur.

There is insufficient data in the literature or in this survey to explain satisfactorily spontaneous uncomplicated nasal hemorrhage in otherwise normal children or young adults. We do not under estimate the possibility that "endogenous factors" may play a rôle in this group, as suggested by Dohlman.[42]

It would appear simpler to consider the following hypotheses, however. Overdrying of the nasal mucosa, *e.g.*, following upper respiratory infection, may cause crusting and scaling of the thin epithelium in Little's area. As a result of local trauma associated with vigorous blowing or picking of the nose, the few cell layers of protecting epithelium may be removed, damaging the superficial vascular plexus. It can be visualized from the plastocene model by Alverdes and from Dohlman's sections, how irregular and incomplete thrombosis of the capillary loops and venous channels would then result in ineffective control of bleeding and predispose to poor healing and recurrent epistaxis.

Post-nasal packing in combination with anterior packing is frequently necessary for control of posterior lateral nasal wall

*The American Heart Association Standards for hypertension were used in our study.

bleeding. As a complication, otitis media, hemotympanum, nasopharyngeal abscess and osteomyelitis[39] may occur. Since the introduction of chemotherapy, and antibiotics, these complications have been less frequent.

The accessibility of the bleeding site determines to a large degree the effectiveness with which hemostasis can be applied. Thus anterior bleeding is controlled much more readily than posterior bleeding. The type of bleeding usually encountered in children is a capillary or venocapillary ooze which is anterior and septal in position, and, therefore, readily controlled. Epistaxis in children is rarely severe enough to cause shock. Brisk bleeding is more common in adults and is posterior in origin, hence more difficult to control than anterior bleeding. As a consequence it is frequently noted that nasal hemorrhage in the adult group may be so severe and so difficult to control as to produce shock and threaten the life of the patient.

It appears that the effectiveness of the anterior nasal pack depends more on mechanical pressure than on the nature of the packing agent. Chemical cauterization usually effectively seals the offending vessel when the bleeding point is readily localized and accessible. This assumes that the bleeding must have been stopped before the cauterizing agent is applied. Electrocauterization was effective in those cases in which brisk bleeding could be visualized but not controlled by simpler means.

Submucous elevation is a highly satisfactory procedure for controlling recurrent, anterior septal bleeding in children. This procedure was successful in every case where packing or cauterizing agents failed to prevent recurrent bleeding. It is a matter of speculation whether the operative intervention in some way alters the vascular supply to Little's area. The elevation of the mucosa may change the supporting framework of the vessel and interrupt some of the blood supply to the anterior nasal septum. Likewise, intractable bleeding over a septal spur may be effectively controlled by a submucous resection.

Ligation of the external carotid artery is a life-saving procedure, and was resorted to as a last measure, in those cases where all other procedures failed to control nasal bleeding.

SUMMARY AND CONCLUSIONS.

1. A brief review of the anatomy and histopathology of the nose, the causal factors of epistaxis, and the proposed methods for controlling nasal hemorrhage are presented.

2. A survey was made of the available records of all cases of epistaxis hospitalized during a 14 year period. The cases were divided arbitrarily into three age groups: *1.* children's group, 48 cases; *2.* young adult group, 42 cases; *3.* older adult group, 46 cases.

3. The incidence of epistaxis was greater among males in the two adult groups. Among children, females were affected more often.

4. The incidence of epistaxis was greatest between the ages of five and 25 years.

5. Epistaxis was present in 50 per cent of children with active rheumatic fever or inactive rheumatic heart disease.

6. Approximately 60 per cent of all epistaxis in older adults occurred in patients with hypertension and generalized vascular sclerosis.

7. In evaluating the site of bleeding it was considered desirable to classfy its origin as anterior or posterior, as well as septal or lateral.

8. Epistaxis in children was always anterior and septal. In the young adult group approximately 80 per cent occurred anteriorly, and was predominantly septal. Twenty per cent of the cases were posterior and lateral.

9. In striking contrast, over half of the older adult nasal bleeding was posterior and lateral in origin. Anterior bleeding in this group was almost always septal.

10. The usual methods of hemostasis for anterior nasal bleeding, *e.g.*, anterior nasal packing, electrocoagulation and chemical cautery, controlled single instances of bleeding but did not prevent recurrent episodes. Recurrent episodes in children were controlled effectively by submucous elevation.

11. A combination of post-nasal and anterior packing was necessary in a higher percentage of posterior bleeding in the older adult group. This was required in a smaller number of the young adult group and was not utilized in any of the children.

12. Otitis media frequently complicated post-nasal packing. This rarely occurred following anterior nasal packing.

13. Ligation of the external carotid artery was occasionally a life-saving procedure in the control of epistaxis.

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THE HUMAN EAR AS AN ANALYZER OF SOUND.

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INTRODUCTION.

The ability of the human ear, under certain conditions, to resolve complex sounds into their component frequencies is well known. This paper describes a method whereby this resolution is quantitatively demonstrated. The method makes use of a complex tone generator in which the harmonics of the fundamental frequency of the complex tone can be cancelled. Subjectively, the ear is able to judge quantitatively when any one of the harmonic components has been eliminated.

Any complex tone may be considered as being made up of a set of pure tones which are harmonics of the fundamental frequency of the complex tone and which have amplitudes which are characteristic of the quality of the sound. For example, we may consider an earphone vibrating so that it is abruptly excited for a certain time (known as the pulse duration), and then abruptly turned off, the whole process being repeated at a given rate (the pulse recurrence frequency). The mathematical treatment or Fourier analysis shows that the acoustic wave produced by the earphone is equivalent to a fundamental pure tone at the pulse recurrence frequency, plus a set of harmonics of this frequency. The amplitudes of the successive harmonics vary periodically at a rate determined

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by the ratio of the pulse duration to the period of the fundamental. Thus, if the pulse is on for one millisecond and off for four milliseconds, mathematical treatment shows that the 5th, 10th, 15th, etc., harmonics are completely absent. There is also a regular decrease in the amplitude of the higher harmonics, so that eventually the high frequencies are present in negligibly small proportions.

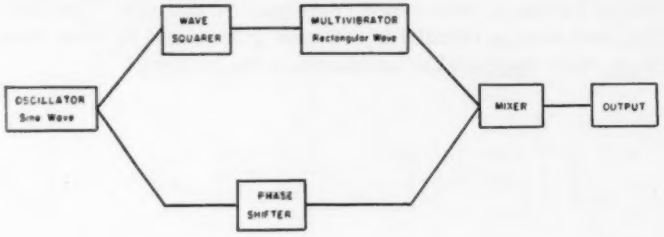

Figure – 1

Block Diagram of Complex Tone Generator

APPARATUS.

Fig. 1 shows the block diagram of an apparatus for generating in an earphone a recetangular wave form of variable height, duration and recurrence rate. The production of pulses of rectangular wave form is accomplished electrically using a multivibrator controlled by an audio frequency oscillator. In addition, a mechanism is provided whereby there can be added to the complex tone any one of the harmonics at arbitrary phase and amplitude.

Even with the finest quality earphone, the pulse is distorted in the transformation from an electrical to an acoustic wave and with a poor quality earphone the acoustic wave produced bears no resemblance to a rectangular pulse. The receiver chosen was a Western Electric Type 711-A. The acoustic output could be examined visually by means of a sound pressure meter consisting of a Western Electric Type 640-A condenser

microphone, a Sanborn Type 43-145 preamplifier and a cathode ray oscilloscope.* The earphone was coupled to the sound pressure meter by a cylindrical cavity, 6 cc. in volume, which is approximately the volume of the external ear canal. At all frequencies above 200 cps. and at all pulse widths used, the sound wave form was rectangular. Using the General Radio Wave Analyzer, Type 736-A, an analysis was made of the electrical output of the sound pressure meter for six pulse widths having a recurrence frequency of 200 cps. The wave structure was as calculated for such pulses, and in most cases 40 or more appreciable harmonics were present.†

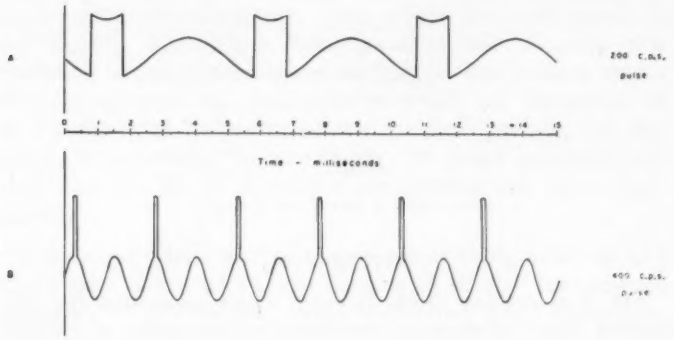

Figure - 2

Fig. 2a shows a pulse five milliseconds in duration at 200 cps. with the fundamental 180° out of phase, and Fig. 2b shows a 400 cps. pulse of one-fourth millisecond duration, with the second harmonic exactly in phase. The complete harmonic analysis of a typical pulse is given in Table 1. The tests were carried out in one of the quiet rooms of the Department of Physics, University of Toronto.

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*The response of this system is flat to 5000 cps. and is down 3 db. at 9000 cps.

†F. E. Terman Radio Engineers Handbook, p. 22. New York and London: McGraw-Hill Book Co., 1943.

TABLE 1.

HARMONIC ANALYSIS OF TYPICAL COMPLEX TONE.

No. of Harmonic	Frequency	Voltage Generated by Condenser Microphone	No. of Harmonic	Frequency	Voltage Generated by Condenser Microphone
	Cycles Per Sec.	Millivolts		Cycles Per Sec.	Millivolts
1	256	100	24	6144	9.4
2	512	100	25	6400	3.8
3	768	78	26	6656	.43
4	1024	37.8	27	6912	2.14
5	1280	5.3	28	7168	2.50
6	1536	16.3	29	7424	2.2
7	1792	28.0	30	7680	1.45
8	2048	28.0	31	7936	.29
9	2304	19.3	32	8192	.72
10	2560	5.7	33	8848	1.47
11	2816	8.0	34	8704	1.94
12	3072	16.4	35	8960	1.46
13	3328	17.8	36	9216	.39
14	3584	12.6	37	9472	.44
15	3840	4.7	38	9728	.54
16	4096	3.9	39	9984	.40
17	4352	7.9	40	10240	.22
18	4608	9.4	41	10496	.10
19	4865	8.2	42	10752	0.00
20	5120	4.1	43	11008	.09
21	5376	1.5	44	11264	.08
22	5632	7.8	45	11520	.02
23	5888	13.2	46	11776	0.00

WAVE STRUCTURE AND PITCH.

On listening to a 400 cps. pulse with a pulse duration of one-fourth millisecond, the complex tone is found to have a reed-like quality and to possess a definite pitch. At first, to most observers, there is no structure to the tone, but on care-

ful listening it is possible to pick out a pure tone of the same pitch as the note and, perhaps, one or more pure tone harmonics.

To emphasize the fundamental component of the complex tone, a sine wave is added to the complex tone at large amplitude and adjusted visually to be 180° out of phase. As the amplitude of the sine wave is reduced, the pure tone component diminishes in intensity, and at one position the fundamental can no longer be heard. If now the added out-of-phase fundamental is suddenly reduced to zero, the pure tone in the rectangular wave becomes very prominent, perhaps even dominating the sound. Thus this technique of canceling the fundamental and then removing the canceling wave focuses attention on the pure tone component in the complex sound and it thereby becomes easy to detect. On listening to the complex sound for some time, the pure tone component gradually becomes more and more difficult to observe and after some time the pulse wave seems to be a single tone again, not pure, but with characteristic pitch and quality. It is worth noting that when the fundamental is made to vanish by cancellation only, the quality of the note changes, the pitch does not seem to change at the same time.

Attention can be drawn to the second harmonic of the sound in a similar way. The audio frequency oscillator is set to twice the fundamental frequency and the intensity of the signal is reduced until the multivibrator functions on every second wave. The pure tone second harmonic can then be added in any phase and any amplitude, permitting the cancellation of the pure tone in the complex sound as described above. The higher harmonics can be heard in exactly the same way. Without this technique of cancellation, it is practically impossible to detect a pure tone component higher than the third or fourth harmonic, but with it the components may be picked out one by one until well past the tenth. Higher harmonics become increasingly difficult to hear (partly because they are present in lesser proportion) as they soon get lost in the complex tone. Components beyond about the twelfth cannot

be heard in the complex tone itself, even just after the canceling pure tone is suddenly removed, but a distinct minimum is heard as the amplitude of the out-of-phase canceling tone is slowly decreased from a large value. In this way it is possible to hear, for example, the twenty-seventh harmonic in a complex tone with a recurrence rate of 200 cps.

QUANTITATIVE DETECTION.

To measure the ability of the ear to detect the presence of a harmonic quantitatively, the amplitude of the complex tone is set so that the pure tone is at some arbitrary level as indicated by the harmonic analyzer. The phase and amplitude controls of the added fundamental are varied (with no refer-

TABLE 2.

THE SUBJECTIVE DETECTION OF HARMONIC TONES PRESENT IN A COMPLEX TONE.

No. of Harmonic	Frequency	Magnitude of Analyzed Voltage Generated by Condenser Microphone	
		Before Cancellation	After Cancellation
	Cps.	Millivolts	Millivolts
1	200	0.30	0.02
2	400	0.28	0.01
3	600	0.30	—0.02
			0.04
4	800	0.23	0.02
			—0.02
			0.02
5	1000	0.12	—0.03
			0.00
			0.01
9	1800	0.08	0.02
			0.03
			0.02

Condition of Test: Pulse duration — 1 millisecond. Recurrence rate — 200 cycles per second.

Note: Harmonics 6, 7 and 8 were not present in the complex tone due to the structure of the acoustic wave.

ence to the oscilloscope) until it is subjectively judged that no fundamental is present. The amplitude of the fundamental actually present is then measured acoustically by the sound pressure meter and the wave analyzer. If increasing the pure tone decreases the indication on the wave analyzer, the judgment was considered to be made with insufficient canceling fundamental and such indications are called negative. The indications are considered positive when increasing the pure tone increases the indication on the wave analyzer. Remainders, due mostly to an inexact adjustment of the phase controls, are not given a sine. Since the pure tone vanishes only at the correct position of both controls, the minimum is very sharp for a proper setting. If one of the controls is in error, the minimum produced by the other does not go to zero, so that the sharpness of the final minimum is the best indication that the balance has been made. Measurements for the higher harmonics are made in the same way. Table 2 shows the readings for a typical set of observations.

CONCLUSION.

These readings show that the ear is able to judge exactly when a given harmonic is absent from a complex tone. In this way the ability of the ear to perform a quantitative structural analysis of a complex sound is illustrated.

DYSPHAGIA AND EXTRAESOPHAGEAL PATHOLOGY.

A Clinical Review of Some of the Important Surgical Lesions from the Endoscopic Viewpoint.*†

V. K. HART, M.D.,
Charlotte, N. C.

Dysphagia is always a symptom which should elicit our closest attention and thorough clinical investigation. The cause may be, and frequently is, some intraesophageal pathology, such as carcinoma, stricture, foreign body or some congenital malformation. Benign growths of the esophagus, in our experience, are rare.

These intraesophageal causes of dyhphagia are not considered in this presentation. Postcricoid carcinoma and tumors of the hypopharynx are also purposely excluded. Likewise, neuromuscular lesions, such as bulbar palsy and myasthenia gravis, together with hysterical and functional dysphagia, are disregarded. I thought it would be more interesting and instructive if we considered some of the extraesophageal lesions that may give rise to dysphagia and with respect to which there have been important surgical advances in recent years, particularly since some of the causes may be overlooked if both esophagoscopy and Roentgen studies are not done; furthermore, some of these conditions may be difficult to differentiate clinically. I refer especially to diaphragmatic herniation, cardiospasm and malignancy of the cardia. The latter two may be confused even by the expert Roentgenologist.

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Last, the symptoms may be quite confusing at times; indeed, diaphragmatic hernia and cardiospasm may simulate coronary and gall bladder disease as pointed out by Vinson,[1] Trueman,[2] Herman and Singer,[3] and Clark.[4]

Pharyngoesophageal Diverticulum. It is logical to begin the discussion anatomically with the pharyngoesophageal diverticulum. This phrase is not accurate in describing what is really a herniation of the mucosa and submucosa of the hypopharynx between the inferior constrictor of the pharynx and cricopharyngeus.[5,6] Such herniations are commonly on the left but may occur on the right or posteriorly in the midline. Harrington[6] states that in some instances the less common median types may manifest themselves below the lower level of the cricopharyngeus. This is a pulsion mechanism in contradistinction to the traction type of true esophageal diverticulum found occasionally at the level of the crossing of the left bronchus.

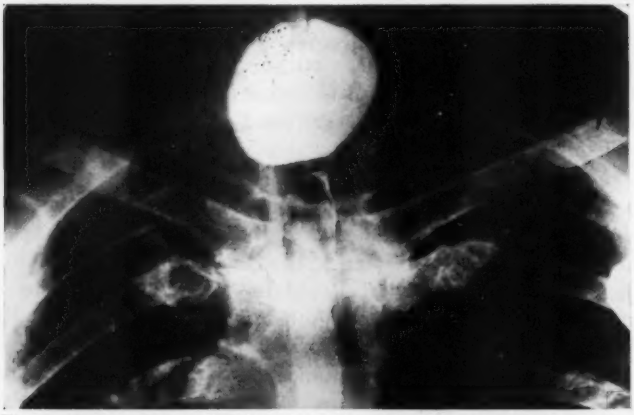

Fig. 1. Case 1. Large diverticulum of the pharyngoesophageal type. The barium-filled sac is plainly shown in this anteroposterior view.

Case 1: Pharyngoesophageal diverticulum. The record of this 54-year-old male is briefly epitomized. He was first seen on June 21, 1946. The chief complaint was difficulty in eating and drinking. His symptoms had begun two years previously. He first noticed that food seemed to stop in the upper food passage. The dysphagia had slowly gotten worse with a 54-pound weight loss. At times, he found it necessary to leave the

table and regurgitate food. On assuming the recumbent position, he had a full feeling related to the upper sternum and which was worse if he turned on his side.

The only findings of clinical significance in his physical and laboratory examinations were those found by X-ray and esophagoscopy. The former showed a large diverticulum typically located in relationship to the upper esophagus. It measured about 5 cm. in diameter.

Esophagoscopy revealed a large blind sac near the cricopharyngeus. At that time, I was not able to definitely identify the opening of the cricopharyngeus. Later, by means of a black silk thread previously swallowed, we were able to identify the opening and enter the esophagus, which showed no pathology. Residual food, of course, was found in the diverticulum. By means of the thread, a feeding tube was placed in the stomach. This allowed us to build up his nutrition. A single-stage operation with the help of Dr. Paul Sanger was done under local anesthesia on Aug. 1, 1946. The patient made an uneventful convalescence and has remained symptom-free since.

Comment: There is no more grateful patient than one relieved of this so-called diverticulum. With the advent of chemotherapy, the single-stage operation, as advocated by Harrington,[6] has come into more general use. In a patient with some contraindication due to other co-existing pathology, the two-stage operation as done by Lahey[5] will probably continue to be useful. At times, he has merely suspended the sac and left it.

In a very large diverticulum, the cricopharyngeal opening may be so displaced by the weight of the diverticulum that the patient cannot even swallow a thread. One of our patients presented such a problem. We had to introduce a shot, to which the thread was attached, under direct endoscopic vision. Even then, we could not thread a feeding tube into position, but had to introduce the tube with the thread as a guide under direct vision with the aid of bronchoscopic forceps.

I feel more secure with a feeding tube in position prior to operation and left there for the immediate postoperative period. This seems a safer procedure for men of limited experience with this condition, such as myself. Harrington[6] does not introduce a tube unless a cervical fistula develops.

As with any obstructive esophageal situation, the frequency of chest complications must be kept in mind. The danger of aspiration of food and saliva is ever present.

Such symptoms may even be misleading. We recently had a patient with a pharyngoesophageal diverticulum, but we also fund a co-existing and advanced carcinoma of the left bronchus. The importance of complete study is thus emphasized.

Superior Mediastinitis. Proceeding downward, anatomically, it is now appropriate to present a case of superior mediastinitis. The clinical history and course were most interesting and are, therefore, abstracted in some detail.

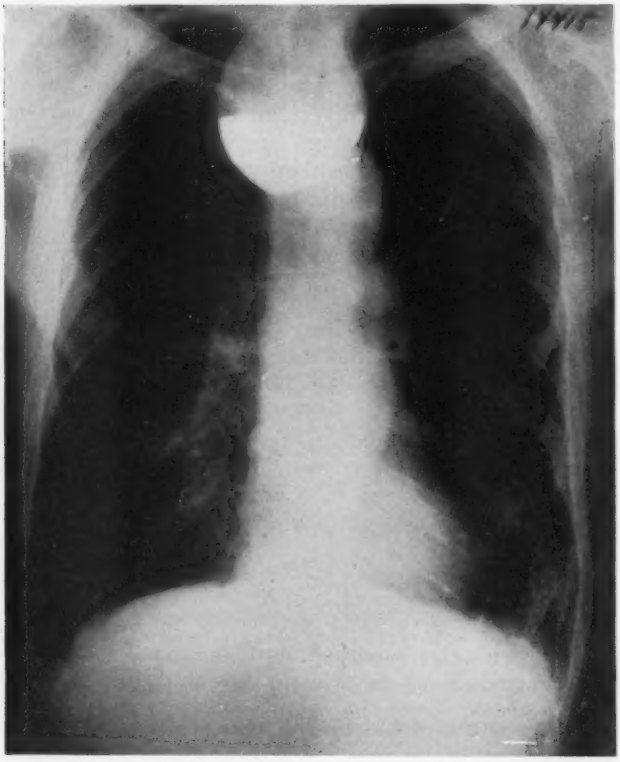

Fig. 2. The X-ray film of another large pharyngoesophageal diverticulum is reproduced. A fluid level of barium is clearly seen in the sac itself.

Case 2: Superior mediastinitis. This patient was a white, married female, 33 years of age. She presented herself on Aug. 15, 1941, complaining of a painful throat. She had been well until 10 days previously. At that time, while eating an overdone homemade biscuit, a piece lodged in her throat. She went to the hospital in her home community where local manipulation was done. She stated the piece of biscuit then went into the esophagus where it lodged. Since then, she had been able to swallow liquids only and had had some fever for the preceding three days.

Examination showed a bilateral infiltration of the neck from the upper level of the thyroid cartilages to the lower neck. She swallowed with great difficulty, followed by strangling. Indirect laryncoscopy showed edema of the hypopharynx and arytenoids. The cords seemed to move poorly. A lateral X-ray film revealed marked swelling of the soft tissues anterior to the cervical spine and in the center of the swelling a pocket of either gas or air.

An esophagoscopy was attempted the same day under local anesthesia. The patient developed immediate dyspnea. I did not attempt to complete the esophagoscopy but proceeded immediately with a tracheotomy under local anesthesia. This was completed without difficulty.

All feeding by mouth was stopped. Glucose and saline were given intravenously. The following day she was given 300 cc. of whole blood by the indirect method. At the time of her transfusion, her blood count was: R. B. C., 4,760,000; W. B. C., 41,500; and hemoglobin, 11.5 gm., or 74.5 per cent. Urinalysis was normal.

On Aug. 16, 1941, endoscopy was done under local anesthesia. The hypopharynx was first exposed with a Jackson laryngoscope. A fluctuating mass, just above the right arytenoid, was noted. This was incised with a laryngeal knife, alligator forceps inserted and foul pus evacuated by suction. A No. 5 esophagoscope was then gently inserted into the esophagus and the upper portion explored. There was marked edema of the mucosa, but no foreign body was seen.

Two days later, this patient became stuporous, cyanotic and the temperature rose to 105.6° (r.). These symptoms were thought to indicate a superior mediastinitis and an immediate drainage of the superior mediastinum by way of the neck approach was done. Light ether anesthesia, supplemented by local novocaine infiltration, was used. Foul-smelling pus was encountered about the upper esophagus and the dissection carried downward into the upper mediastinum. Drains were placed and the wound closed.

She developed marked respiratory depression immediately afterwards, requiring oxygen, artificial respiration and stimulants. Five hundred cc. of blood had been started by vein as the operation was begun. Following this, 500 cc. of 5 per cent glucose and saline were given intravenously. She regained consciousness a few hours later. At this time, a Levine feeding tube was passed through the nose into the stomach.

She was given another transfusion the following day. She steadily improved, and she was extubated and discharged from the hospital on Aug. 28, 1941. She still had some slight discharge from the neck wound, but this healed uneventfully within two weeks.

Comment: I feel that her infection was probably caused by trauma from the biscuit and that it probably began as a retropharyngeal abscess. Her subsequent course, after drainage of the hypopharynx, can be explained only by a descending infection into the mediastinum. The patient's recovery, with drainage of this area, leave little doubt as to the mediastinitis.

In our experience, other extraesophageal lesions producing dysphagia between the cricopharyngeus and the diaphragmatic hiatus are quite uncommon. Some of the causes we have encountered in this area have been metastatic glands from carcinoma of the breast and marked curvature of the spine. Vinson[7] also mentions tumors of the thyroid, acute thyroiditis and aneurysm. Mosher[8] recorded cervical exostoses as a cause of dysphagia.

Hiatal Hernia. The next important extraesophageal lesion as we go downward, and possibly one of the most frequently overlooked, is the hiatal hernia. The first case found at operation at the Mayo Clinic, according to Harrington,[9] was in 1908, and the first diagnosis by X-ray at the same clinic was in 1921.

It might be well to review briefly the types of diaphragmatic hernia. Harrington[10] divided them into traumatic and nontraumatic. The nontraumatic may be congenital or acquired. If congenital (still quoting Harrington), the sites in probable order of frequency are *a.* through the hiatus pleuroperitonalis (foramen of Bochdalek), *b.* through the dome, *c.* through the esophageal hiatus, *d.* through the foramen of Morgagni, *e.* through the gap left by the absence of the left diaphragm.

Embyryologically, Dorsey[11] states the most common type of diaphragmatic hernia is through the foramen of Bolchdalek. He reports two infants in whom surgical repair was successfully done and states that the child with such may die at birth from asphyxia or aspiration pneumonia. Surprisingly, he places hiatal hernia as the second most common

type, because McNealy[12] and his confreres report that it constitutes more than two-thirds of all nontraumatic diaphragmatic hernias.

Harrington's[9] figures show that practically all of his patients operated upon for hiatal hernias were of the paraesophageal type. He describes two subgroups: *1.* The lower esophagus is not above the diaphragm. *2* The lower esophagus is above the level of the diaphragm. He believes that a true congenitally short esophagus is rare, because he found only five cases at operation in his large series, four of which could be corrected with the stomach below the diaphragm.

I was rather surprised at this low incidence of congenitally short esophagus. I found in our files from 1941 to date, 17 cases in which a diagnosis of diaphragmatic herniation had been made. Two cases were diagnosed by X-ray only, but the X-ray reports did not clearly differentiate between the short esophagus and the paraesophageal type. Fourteen other cases done by myself were all diagnosed as herniations at esophagoscopy. My diagnosis was confirmed by subsequent X-ray studies in nine of the 14 patients, but again the X-ray reports were indefinite as to whether the patient had a paraesophageal type or a congenitally short esophagus. By reviewing what films I could find, three were found showing a congenitally short esophagus.

Roentgen studies did not confirm the esophagoscopic diagnosis in three patients. Two other such patients had no follow-up X-ray studies.

One patient clearly showed the herniation on the X-ray film, together with a stricture above. Esophagoscopy, however, demonstrated only the stricture as the 'scope could not be gotten safely beyond the obstruction, even after endoscopic dilatation; however, whether part of the stomach is through the hiatus because of a congenitally short esophagus, or because of some congenital deficiency in the hiatus, or because of injury, is largely academic to us as endoscopists. The important thing is the diagnosis.

In that connection, by far the most common complaint was difficulty in swallowing. A few complained of burning beneath the sternum and one, of pain. Frequently there was a history of periodic difficulty in swallowing, with or without vomiting, extending back over a period of years. Only one of our patients had had bleeding.[13]

Several times I have had patients come in completely obstructed with a bolus of food. Diaphragmatic herniation was not suspected until we found the food mass in the herniation. This was also reported by Clerf and Manges.[14] I should like to recapitulate briefly such a case and two other typical cases without food impaction.

Fig. 3. Case 2. Lateral X-ray view of neck showing marked bulging of the soft tissues anterior to the cervical spine. There is also a significant pocket of gas or air showing in the swelling itself. This patient later developed a mediastinitis of the superior mediastinum.

Case 3: Hiatal hernia with superimposed food impaction. This woman, 86 years of age, was first seen by a gastroenterologist on Dec. 13, 1945, because of inability to swallow. This had come on suddenly, 48 hours previously, while eating. The patient was studied with barium, a diagnosis of foreign body made, and the patient was referred to me for esophagoscopy. This was done under local anesthesia, using an 8 x 45 Jackson esophagoscope. At 33 cm. from the upper gum margin, a large,

impacted food mass was found. Following its removal, it was found that the 'scope met firm resistance at 36 cm. with no evidence of intra-esophageal tumor. There was no stricture, and I thought I could see stomach mucosa beyond. The esophagoscopy was discontinued and the patient sent back to the referring physician with a diagnosis of diaphragmatic herniation with secondary impaction of food.

This diagnosis was confirmed by subsequent X-ray studies; moreover, questioning of the family brought out a history of periodic esophageal obstruction for years. Several times something had been dislodged with a stomach tube by a physician of her home community.

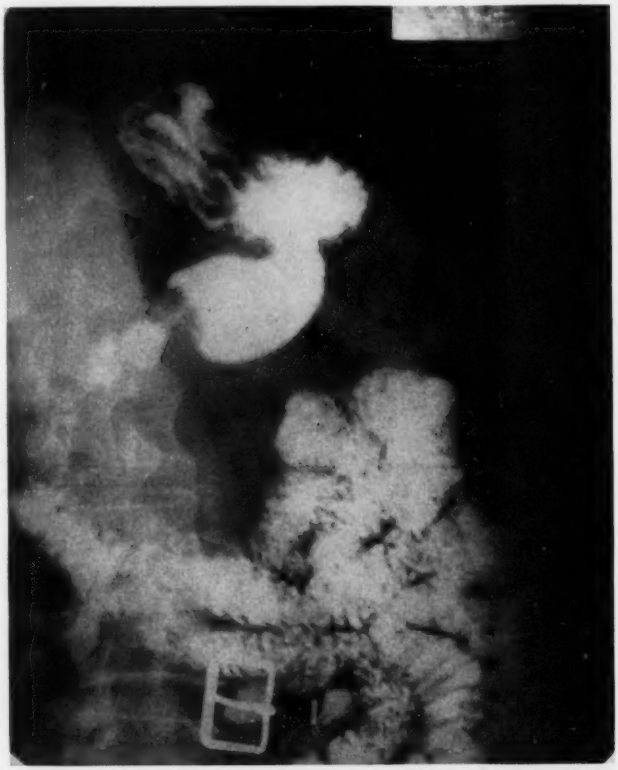

Fig. 4. Case 4. Oblique view of diaphragmatic herniation. The striations of the barium-coated stomach rugae are clearly seen above the level of the diaphragm. The esophagus and the esophagogastric junction show poorly in this film. The lower borders of the esophagus have, therefore, been outlined with ink for greater clarity. The Roentgenologist thought this was probably a herniation with a congenitally short esophagus.

Case 4: Hiatal herniation with congenitally short esophagus. This patient, 62 years of age, was referred by his family physician on Jan. 20, 1944, for an esophagoscopy. His chief complaint was difficulty in swallowing. He had had periodic trouble for two years, but denied trouble in earlier life. There was no history of ingestion of a caustic. Esophagoscopy was done under local anesthesia. An 8 x 45 Jackson esophagoscope was passed. The esophagus showed no pathology but stomach mucosa was encountered at 37 cm. from the upper gum margin. The 'scope could not be forced into the stomach without undue trauma. There was a small, brownish, yellow mass within the folds of the cardia and apparently attached to the left wall. It did not impress me as malignant but small specimens were removed with punch forceps. (Microscopic examination later showed these to be food.) The esopha-

Fig. 5. Case 5. This anteroposterior view reveals a large diaphragmatic herniation. The greater redundancy of the esophagus in this case is indicative of the paraesophageal type.

goscopy was then discontinued and a diagnosis made of hiatal hernia, probably with a congenitally short esophagus. This diagnosis was sustained by later Roentgen studies.

Case 5: Paraesophageal hiatal hernia. A white male, aged 60 years, was admitted to the hospital on May 29, 1945, with the complaint of of inability to swallow. He stated that he had been unable to retain anything for three days. There had been periodic difficulty in swallowing for 15 years. It was apt to come on suddenly while eating. He would then vomit and have no further trouble until the next attack. The symptoms this time had lasted longer than with any previous attack.

Esophagoscopy was done under local anesthesia. An 8 x 45 Jackson esophagoscope was passed. Just beyond 35 cm. from the upper teeth, what was apparently stomach mucosa was encountered. I was not able to get the 'scope below a level of 37 cm. without unsafe force, and I could not get a bougie into the stomach with reasonable manipulation. The esophagoscopy was then discontinued. A diagnosis of diaphragmatic herniation was made with probably a congenitally short esophagus. X-ray study showed the hiatal herniation of a paraesophageal type. The patient was able to swallow following the instrumentation. Four months later he returned with a complaint of not being able to swallow even water. His obstructive symptoms had been going on for two days. An esophagoscopy again verified the herniation at about 36 cm. from the upper incisor teeth. A moderate sized dilator was passed. The following morning he was able to swallow quite well.

Comment: I am not always sure, as illustrated by the last case, whether the herniation, as seen through the esophagoscope, is associated with a short esophagus or one of normal length. Theoretically, it should be relatively easy by accurate measurements to make the differential diagnosis; nevertheless, I cannot always make the distinction. Harrington[9] states that the abdominal esophagus is only 0.5 to 1.5 cm. long; therefore, any redundancy could easily be taken up by pressure of the 'scope on the contralateral wall. The best differentiation is by X-ray.

Most of my patients have done very well on a dietary regime of small meals at frequent intervals rather than large feedings. I have been very conservative in dilating these cases. It would seem unwise unless they have obstructive symptoms, and even then they should be done with great prudence. Some outstanding men, such as Vinson,[7] use dilatation in selected cases. The death of only one patient with a herniation has, nevertheless, made me quite conservative; particularly since she died of a mediastinitis following uncomplicated esophagoscopy without any dilatation. Older patients with an esophagitis of long standing should especially inspire caution.

The most dramatic results have come from surgery. Either the abdominal or the transthoracic approach may be used. Both have their advocates. Detailed discussion of this aspect is not pertinent.

The ulcer of the esophagus near the esophagogastric junction, which occasionally occurs, should be mentioned. This may be from the pressure of the hernia itself or the irritating effects of the gastric contents.[9] This in turn may give rise to stricture with obstructive symptoms. Such symptoms may also be due to a concomitant cardiospasm. Two of Harrington's[10] patients continued to have cardiospasm after operation.

A second case of herniation with stricture above the cardia (one other case previously mentioned) was observed. This required dilatation through the esophagoscope. Later he was dilated by a dilator on a swallowed thread, but got quite a reaction with fever. He promptly recovered with hospital management and chemotherapy. Since then I've been quite willing to let well enough alone, as long as the patient has no marked obstructive symptoms.

Finally, hiatal hernia and cardiospasm may occur secondary to the sclerosis of acrosclerosis and scleroderma. The herniation occurs as a result of secondary shortening of the esophagus, and the achalasia possibly because of changes in the esophageal wall which could interfere with the nerve supply. These facts are brought out in a very excellent paper by Olsen[15] and his associates. I have never seen such a case.

Cardiospasm and Carcinoma of the Cardia. As we end our journey down the esophagus, it is appropriate to consider these two extraesophageal lesions together. I say this because it is not always easy in the early stages to differentiate by either X-ray or esophagoscopy the early tumor of the cardia and cardiospasm. I am sure that all endoscopists agree that every case with a tentative X-ray diagnosis of achalasia should be esophagoscoped before any dilatation is done.

Not only is tumor of the cardia confused with cardiospasm but at times even with hiatal hernia. The following cases illustrate some of the problems encountered.

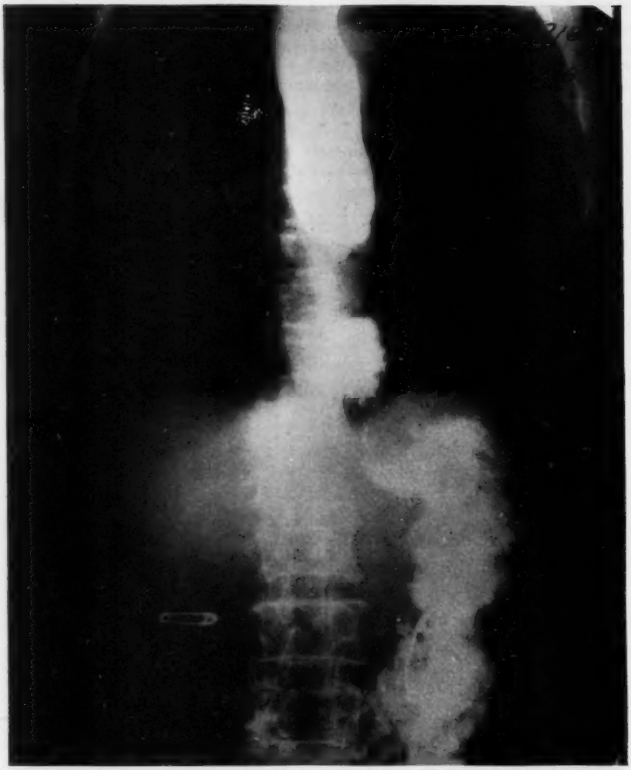

Fig. 6. Both an esophageal stricture (A) and a diaphragmatic herniation (B) are shown in this photograph. Such a stricture is probably the end-result of esophageal ulceration secondary to the herniation.

Case 6: Carcinoma of the Cardia Suggesting Esophagoscopically Hiatal Hernia and Cardiospasm by X-ray. The patient was 69 years of age and first seen on Oct. 29, 1944. Her chief complaint was difficulty in swallowing. Her present trouble had begun three months previously when she began to regurgitate food. This became worse, until at the time of admission she could swallow and retain very little food. She had lost 40 pounds in weight. Her daughters stated that for a good many years she had had one or two attacks a year of pain and indigestion, followed by vomiting, which would relieve her symptoms. A recent X-ray study had shown obstruction at the cardia, and the question arose as to whether this was cardiospasm.

Esophagoscopy was done under local anesthesia, using an 8 x 45 Jackson esophagoscope. Some retained secretion and liquid were aspirated.

The esophagus was normal until what was thought to be stomach mucosa was encountered between 36 and 37 cm. from the upper gum margin. Resistance was firm and no dilator could be safely passed. No abnormal tissue was seen. I thought she probably had a congenitally short esophagus with a hiatal hernia and the esophagoscopy was discontinued.

X-ray studies were done by a very excellent roentgenologist and failed to reveal any herniation or definite evidence of tumor. A diagnosis of probable cardiospasm with esophagitis was reported. It was subsequently found that a Mosher air bag could not be introduced into the stomach.

The esophagoscopy was then repeated. At 35 cm. from the upper gum margin, there was encountered what was either edematous esophageal

Fig. 7. Case 6. The obstruction of the lower esophagus shown here was diagnosed radiographically as cardiospasm. Esophagoscopically, there was some suggestion of diaphragmatic herniation. An advanced carcinoma of the cardia was demonstrated surgically.

mucous membrane due to an intramural or periesophageal mass, or stomach mucosa. I got the definite endoscopic impression that there was a mass below the obstruction in the periesophageal tissues.

The patient was losing ground and vomiting almost constantly. She was, therefore, referred for surgical exploration. This was done nine days after admission, and an extensive carcinoma of the stomach found, which had already ruptured but had been walled off by the omentum. It was obvious that there was nothing that could be done surgically.

Case 7: Carcinoma of the Cardia Suggesting Roentgenographically Cardiospasm. This white male, age 63 years, was referred for esophagoscopy on April 13, 1943. He complained of inability to eat and had had progressive symptoms for several months. The roentgenologist reported a cardiac obstruction suggesting cardiospasm.

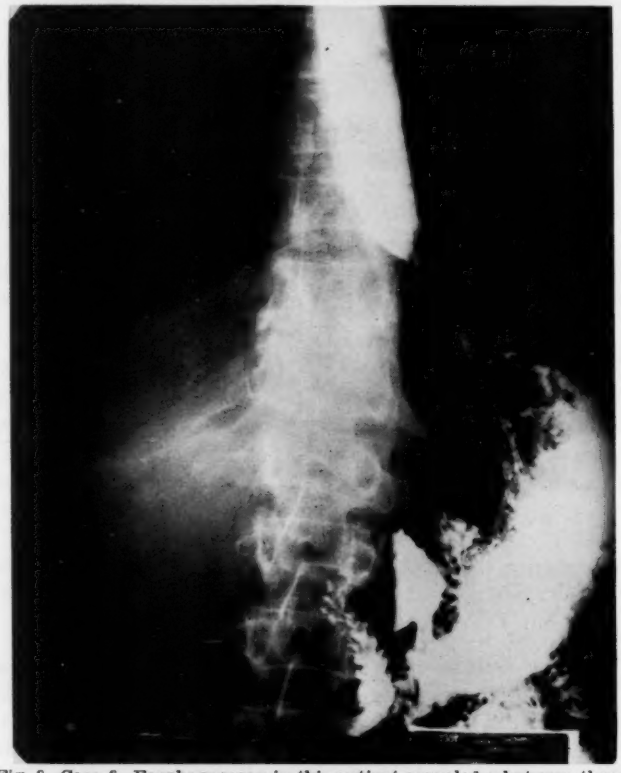

Fig. 8. Case 8. Esophagoscopy in this patient revealed what was thought to be cardiospasm. A later gastroscopic examination on the insistence of the radiologist revealed probable carcinoma of the cardia, which was sustained by the histopathologic report of the biopsy.

Esophagoscopy was done under local anesthesia. An 8 x 50 Jackson esophagoscope was passed. At the cardia, encountered at a normal distance, was found a small, bleeding, indurated mass, attached to the left wall. Biopsy was done and the esophagoscopy discontinued. The pathologic report was adenocarcinoma.

Case 8: Carcinoma of the Cardia Almost Missed by Improper Visualization. This 53-year-old male was referred to me by an internist for esophagoscopy. He had had dysphagia and the X-ray findings showed an obstruction at the cardia suggestive of tumor. I did an esophagoscopy, using an 8 x 45 Jackson 'scope, with negative findings. More X-ray studies were done and reported as showing a lesion in the upper stomach. The esophagoscopy was repeated under local anesthesia on March 10, 1948. This time I used an 8 x 50 'scope and a lesion on the right lateral wall of the stomach, at 46 cm. from the upper gum margin, was easily visualized and biopsied. The pathologic report was adenocarcinoma.

An extensive resection was done with an esophagogastrostomy by the consulting chest surgeon; nevertheless, he died of extensive abdominal metastases six months later.

Comment: The endoscopist should be very suspicious of a malignancy at the cardia if one or more of the following conditions are encountered: First, firm resistance to the introduction of the esophagoscope into the stomach. Especially if pain is registered by the patient. This obstructive phenomenon is quite different from that of cardiospasm as emphasized by Allison.[16]

Second, one should be wary of tumor if a dilating bag cannot be safely introduced into the stomach on a previously swallowed thread. Even if the bag can be introduced, further suspicion should be aroused if a poorly yielding wide band of resistance is noted fluoroscopically, rather than the usual constricting ring seen at the cardia in cardiospasm. There may be even abnormal resistance to the withdrawal of the bag in a malignancy of the cardia. Such cases, though biopsy has not been feasible, or, if done, negative, should suggest surgical exploration. Carcinoma of the cardia will often be found.

Last, the failure of a supposed cardiospasm without any angulation of the esophagus to respond satisfactorily to dilatation bespeaks re-examination of the patient for an unrecognized malignancy. I have just had such a patient. Using a 50 cm. esophagoscope in the second esophagoscopy, an adenocarcinoma of the cardia was readily visualized and proven

by biopsy, although the man was only in his early thirties; moreover, the failure to use the longer instrument caused me to miss the lesion on the first examination in one other case (Case 8).

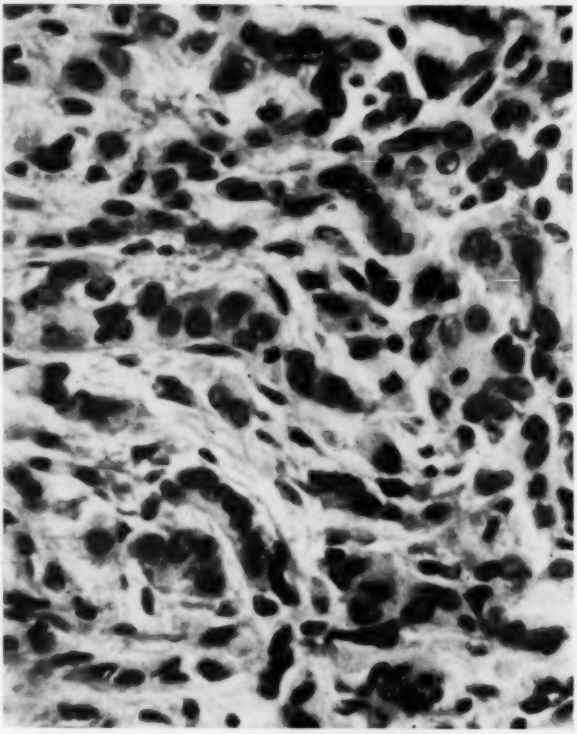

Fig. 9. Case 8. High power magnification showing infiltration with strands of pleomorphic, but mostly cuboidal epithelial cells, the adenomatous character of which is only suggestive. The cells, however, do not bear resemblance to squamous elements. Pathologic diagnosis (Dr. Paul Kimmelstiel): adenocarcinoma.

Vinson[1] makes an important point in the differential diagnosis of cardiospasm. He states it is the only disease which produces definite angulation and dilatation.

This so-called sigmoid type of cardiospasm, which often responds poorly to dilatation, is now being treated successfully by both transabdominal and transthoracic surgery.[17-19] Likewise, new hope is now being offered by thoracic surgery to patients with carcinoma of the cardia and esophagus.[20-24] It is not within the scope of this paper to review this literature, but it is our function as endoscopists to know that this help is now available for these patients.

Summary: The more important extraesophageal lesions causing dysphagia have been discussed with the citation of illustrative cases. They were presented in the order of their anatomical sequence, *viz.*, pharyngoesophageal diverticulum, superior mediastinitis, hiatal hernia and carcinoma of the cardia. Cardiospasm was included only from the standpoint of surgical treatment and differential diagnosis. Emphasis was placed on the endoscopic aspects which concerned diagnosis and management. It is felt that hiatal hernia and carcinoma of the cardia are probably more frequent than usually realized. Attention was called to the recent surgical advances in their treatment.

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CONTINUOUS PHARYNGEAL SUCTION DURING TONSILLECTOMY UNDER LOCAL ANESTHESIA.*

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INTRODUCTION.

When a tonsillectomy is performed under local anesthesia, particularly when the patient is in a sitting or semirecumbent position, several things may or do happen which are undesirable. The patient may become faint and require at least a temporary placement in a recumbent position. Saliva, pus and blood may be aspirated into the tracheobronchial tree, with the danger of an ensuing pulmonary infection.[1,2] The aspirated material may also choke and alarm the patient, while the accompanying cough scatters blood annoyingly about. Even when this does not occur, the accumulation of blood may obscure the operative field and interrupt the operation until the blood can be expectorated.

In an effort to overcome these objectionable features, it has been found that by placing the patient in a certain dorsal recumbent position and by combining this with a method of continuous pharyngeal suction, a satisfactory answer to the problem is obtained. This procedure will be described and is the object of this presentation.

In the development of this procedure continuous suction and the recumbent position were employed from the beginning. These methods appeared most likely to lead to success and each depended upon the other. At first, various modifications

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of the commonly used Yankauer tonsil suction tube were employed, either alone or combined with a metal tongue depressor. Similar suction tubes have been devised by Moore,[3] Myerson,[4] Hunt[5] and others for use primarily in tonsillectomy under general anesthesia. These suction tubes must be held by an assistant, and very often they become displaced from the position where suction is needed. They also tend to get in the way of the surgeon at times. Though their use is more satisfactory than operating without suction and though they have been used for several years, an improved technique was desirable.

A real advance was made in 1945 with the employment of a catheter with multiple perforations near its tip. This was inserted through an anesthetized nasal passage into the oropharynx. Its use was a decided improvement over the oral suction tubes previously employed. Various sizes of catheters and perforations were used until the most satisfactory combination was found. A further refinement in the method resulted when two such catheters were used, because occasionally one tube tends to become partially obstructed by blood clots.

TECHNIQUE.

A. Preparation of the Patient.

Preparation of the patient actually begins when tonsillectomy is advised. Local anesthesia is suggested as the choice of anesthetic if the patient is not too nervous or does not possess an excessive gag reflex. Quicker recovery under this form of anesthesia is pointed out to the patient. Other advantages are: the relaxing effect of adequate sedation and of the recumbent position, and freedom from gagging, choking or expectoration by using suction "which removes saliva and any blood that may run into the throat."

While in surgery the patient is continually assured by proper "vocal" anesthesia and is impressed with the importance of regular breathing and of the avoidance of swallowing.

Preoperative Medication. Adequate sedation and analgesia are both most important. While the preoperative medication for each patient is individualized, the average adult patient receives nembutal* grs. iii orally, one and one-half hours before surgery, then morphine sulphate grs. ¼ with atropine sulphate grs. 1/150 hypodermically, one hour before surgery. Morphine relaxes the stomach and increases the tone of the pylorus, thereby delaying passage of stomach contents. For this reason nembutal, when given orally, is administered in advance of morphine to insure adequate absorption of the barbiturate in the small intestine. If sedation is not sufficient when the patient reaches the operating room, additional medication should be given intravenously. This is seldom necessary, however. Scopolamine is preferred to atropine for some patients because it enhances the degree of hypnosis. The smaller doses are used, otherwise proper cooperation of the patient may not be preserved.

Recumbent Position. The operating table top is put in the horizontal plane. Then a moderately firm bed pillow is placed under the patient's back and shoulders, but not under the neck. This arrangement permits moderate extension of the head and neck so that the posterior pharyngeal wall slopes downward from the larynx to the nasopharynx (see Fig. 1).

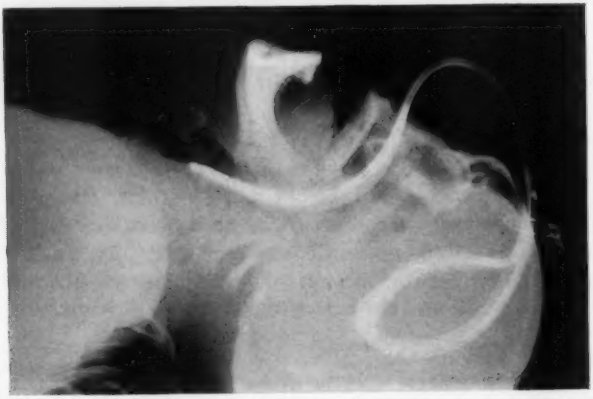

Fig. 1. The downward slope from the larynx to the nasopharynx is shown in this head-extended position.

*Sodium pentobarbital.

This position reduces the chances of aspirating blood. The head extended position is not so protective as the Trendelenberg or Rose positions as shown by Hara[6] in cases of general anesthesia, but, nevertheless, it is quite effective; furthermore, under local anesthesia, the patient is much more comfortable in the head-extended position than in one of the Trendelenberg positions.

A review of the literature on this subject reveals that Bettington[7] also advocated the head-extended recumbent position in performing tonsillectomy under local anesthesia. No mention of suction was made, but possibly it was employed in some form.

Topical Anesthesia (by spray). About 10 minutes before surgery the patient's nose and throat are sprayed with a solution comprised of three parts of 2 per cent pontocaine* and one part of 1 per cent neosynephrine.† If the patient says "ah" as this is being done, the spray is less liable to enter the larynx, and the tongue drops to the floor of the mouth, permitting the spray to strike the posterior pharyngeal tissues. This spraying is repeated five minutes before surgery.

Draping. A sterile body drape, which is split at the neck, is applied. The patient is instructed to raise his head while the tails of the drape are barely overlapped under the head, and next a large sterile towel is placed where the head will be when it is lowered. The ends of the towel are then folded across the forehead and firmly fixed with a towel clip. The head towel should be tight enough not to slip, as the suction tubes are to be fixed to it later.

Topical Anesthesia (with cotton applicator). A metal cotton applicator is used to swab the nose and pharynx with the pontocaine-neosynephrine solution. The applicator is passed through the middle meatus and nasopharynx into the oropharynx and there swabbed from side to side. It is drawn out through the inferior meatus of the nose. This procedure is repeated through each nasal passage a few times until anesthesia is complete (see Fig. 2).

*Tetracaine hydrochloride U.S.P.
†Phenylephrine hydrochloride.

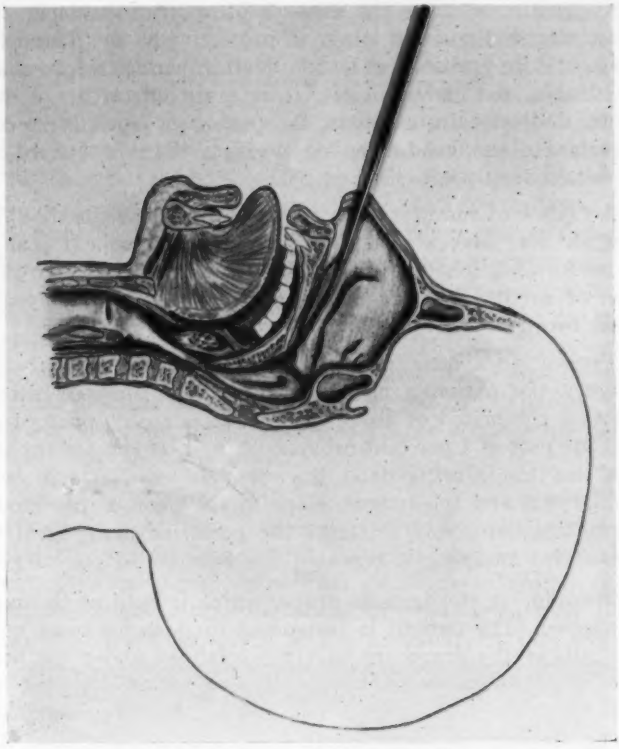

Fig. 2. The metal cotton applicator used to swab the nose and pharynx is about seven inches long with the distal three inches curved in a 90 degree arc.

The lips are anointed with vaseline, and then with an applicator similar to that used in the nose, the mouth and pharynx are swabbed with the same local anesthetic. The areas covered are the tongue, down to its base, the hard and soft palate, the tonsils and their pillars, and the posterior pharyngeal wall down towards the larynx. Applications are repeated until the gag reflex has completely disappeared. Unless this precautionary technique is followed, the suction tubes or surgical procedure will cause gagging and the method will fail.

Suction. Quarter-inch suction tubing is fixed by a towel clip to the top of the table just to the left of the patient's head. The tubing is fitted with a pressure regulating clamp and a quarter-inch glass "Y" tube.

The suction tubes* consist of two 14F male red rubber catheters which have multiple perforations in their tips (see Fig. 3).

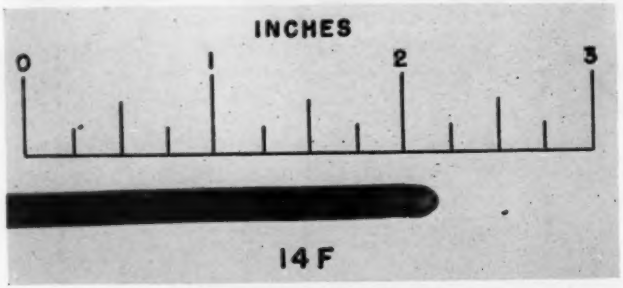

Fig. 3. Tip of a nasal suction tube.

The tip of one of the suction tubes is dipped in vegetable jelly and inserted through the nose into the nasopharynx. This process is repeated with the other tube. The nasopharynx is entered most easily by following the curved path offered by the middle meati; however, if there is an obstruction in the nose, other pathways must be used (see Fig. 4).

*The tubes can be easily made or they may be obtained from the Clay-Adams Co., 141 East 25th Street, New York 10, N. Y. Single eye catheters are selected. Those of natural red rubber seem to hold their shape better than those of synthetic rubber. The tip is grooved around its circumference just back of the rounded portion, with a five-eighths inch double cut Joe Dandy dental separating disc using a straight dental handpiece and machine. The portion just back of this is thinned for about one-eighth inch with the same equipment, so that a mushroom-like cork results. This is severed from the tip, and then the eye of the catheter is cut away. Rubber cement is applied to the inside of the end of the catheter and to the neck of the cork, and the two are cemented together. The rounded tip prevents trauma to the tissues as the catheters are introduced into the nose and pharynx. An opening in the end of the tip is undesirable as it may catch the tip of the epiglottis during suction.

Four openings in the catheter are made in line, one-quarter inch apart, starting just proximal to the end of the rubber cork. A No. 702 crosscut tapered fissured dental burr (straight hand piece) is used to make these openings. They are carried through to the other side to make eight such openings. The burr is passed back and forth to ream out the openings until they are about one-sixteenth inch in diameter. Then at right angles to these holes, two sets of holes are made at the same interval but staggered between the two sets of four holes, resulting in a total of 14 perforations in the tip portion of a suction tube.

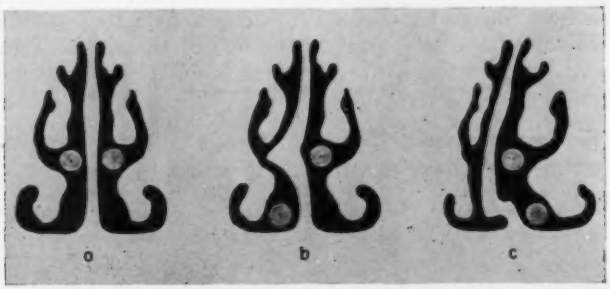

Fig. 4. (a) Suction tubes are usually passed through the middle meati. (b) If one middle meatus is obstructed, the inferior meatal path is followed. (c) If one nasal passage is obstructed, both tubes are passed through the other side of the nose.

Using a tongue depressor, the posterior pharyngeal wall is exposed and the suction tips adjusted so that the top perforation of each is just below the tip of the uvula (see Fig. 5).

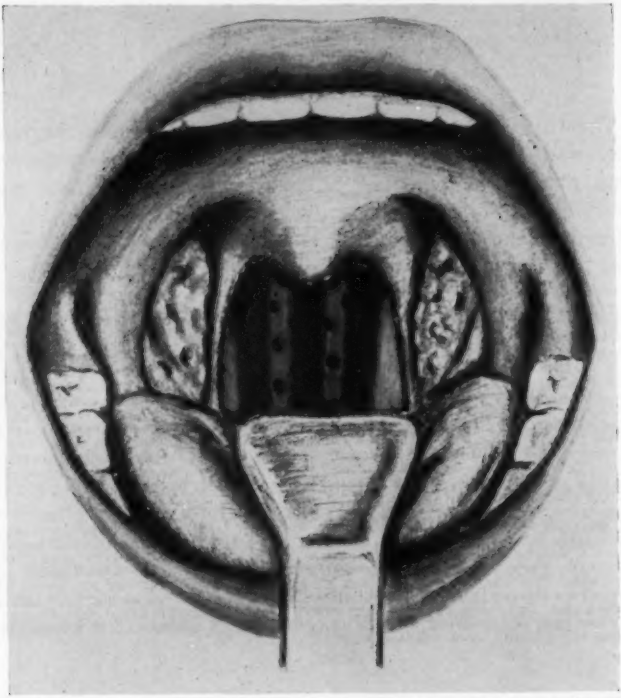

Fig. 5. The tips of the suction tubes are in position. Suction of the uvula is avoided by keeping the perforations of the tubes below it.

The portions of the tubes extending from the nose are carried up to the forehead and are fixed to the head towel by a safety pin. After picking up a piece of the towel, the pin is passed over the tubes to engage the towel again in such a manner as to apply enough pressure to the tubes to keep them from slipping. The bell ends of the tubes are then attached to the arms of the glass "Y" tube. Suction is turned on, and its pressure is regulated by the clamp on the large tubing (see Fig. 6).

Fig. 6. Nasal suction tubes and their connections are shown above. Note particularly the safety pin fixation to the head towel. Between this fixed point and that of the towel clip, an "S" shaped course of the tubing permits some rotation of the head without a pull on the tubes.

Infiltration Anesthesia. Any technique desired by the surgeon may be employed.

B. The Operation.

Here again, the surgeon may use the technique of his choice. It has been found helpful, after one tonsil has been removed, to insert a string sponge into the fossa to control bleeding while the other tonsil is being removed. Some may prefer to ligate any bleeding vessels first. Before proceeding with the

other tonsil, it is also advisable to remove any large stringy clots that may have collected about the perforations of the tubes.

A Davis mouth gag was formerly used to keep the mouth open and the tongue out of the way. But it has since been abandoned for a Weder or similar metal tongue depressor in cases where an unruly tongue must be controlled. Patients prefer these to the Davis type gag because they permit intermittent relaxation of the jaw muscles.

At the conclusion of the operation, the suction tubes are removed slowly through the nasopharynx so as to remove any blood that may have accumulated.

When the patient is returned to his room, he is instructed to lie on one side, and not to use a head pillow for several hours. This position tends to prevent aspiration of any blood.

SUMMARY.

In the foregoing presentation a method of continuous pharyngeal suction, combined with the placing of the patient in a certain dorsal recumbent position, has been described for use in performing tonsillectomy under local anesthesia. A personal conclusion has been reached that by the use of this technique the operation is rendered safer and the inconveniences are reduced for both patient and surgeon.

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THE RELATIONSHIP OF SWIMMING AND DIVING TO SINUSITIS AND HEARING LOSS.*

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INTRODUCTION.

The purpose of this paper is to record data collected in order to determine the relationship of swimming and diving to sinusitis and hearing loss. Two things especially aroused the author's interest in this: *1.* a friend once on a college diving team stated that most of his team mates were deaf, and *2.* a nearby pool filled the office every summer with sinus and ear cases. (Let it be said now that the friend was probably in error, and, second, that the pool referred to was later proven to be greatly contaminated.

The relationship between swimming and diving and sinusitis and hearing loss, or upper respiratory infection, has existed and has been recognized for years. Cobb,[1] in 1908, writing on the "Menace of the Swimming Tank," stated, "Ethmoiditis and otitis media which apparently were caused directly by diving in a swimming tank — occur so often — and are often so serious, not alone in regard to the hearing, but even to the life of the patient. . . ." Mosher[2] has said "our worst cases of frontal sinus infection and osteomyelitis have followed swimming." Taylor,[3] in speaking of public bathing places, stated "aural infections — which can be attributed directly to the sports of swimming and diving." Mackenzie Brown[4] and many others, too many to enumerate, have reported similar instances.

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Various reasons have been advanced to explain why swimming and diving may cause upper respiratory disease. Contaminated water, water containing irritants, or the presence of already existing disease in the swimmer are some of these. Taylor[5] and Taylor and Dryenforth[6] have shown that the body temperature is lowered and that there are blood and vascular changes after the prolonged chilling due to swimming. Man's lack of adaptation to immersion in water as compared to that of aquatic animals has been clearly shown.[7] These animals are able to close their anterior nares and their external auditory canals, thus affording protection from the destructive action of water. Aquatic animals are also well insulated, while man is not. In addition to the above factors predisposing to upper respiratory infection, it is known that solutions which are either hyper- or hypotonic have a detrimental effect upon the mucous membranes.

METHOD OF STUDY.

It was felt that the best way to approach this subject would be to contact the swimmers and divers themselves. To do this, a number of sources were employed. The first and most important source was that of the colleges and universities throughout the country. The athletic director or swimming coach of each of these was sent a letter explaining the nature of the study and asked if he would furnish a list of the former swimmers and divers of that college. Many (48) expressed interest in the undertaking and cooperated fully. Other sources were the amateur athletic union, theatrical booking agencies for professional swimmers, and leads given by the swimmers themselves. To all of these swimmers was sent a form letter addressed individually. It read as follows:

Dear Mr. Smith,

I am conducting an investigation for a scientific (medical) report of the effect (if any) of swimming and diving upon the hearing and sinuses. One of the most important aspects of such a study would be the opinions of the swimmers themselves.

Your name has been given me by the athletic director of your college, who is also interested in the results obtained. If you will fill in and return the enclosed post card your cooperation will be greatly appreciated. Feel free to make any additional comments.

If you are able to come to the above address or to the department of prevention of deafness clinic at the Jefferson Medical College Hospital, we would be glad to examine you (no charge).

Sincerely yours,

Enclosed with the letter was a self-addressed and stamped post card. This post card had spaces where the person could state whether he was a swimmer or a diver, and whether he had continued this activity since his college days. There were spaces to check the presence of hearing loss, and whether he thought this was due to swimming and diving, not due to it, or that he was unaware of the cause. There was a space to record tinnitus. He then checked the presence or absence and severity of sinusitis and again its relationship to swimming and diving. There was a place for comments, the swimmer's class and university.

A control group was also established. This, too, was mostly college alumni. Their names were furnished by the secretary of the University of Pennsylvania. They were graded as to age so as to correspond as nearly as possible with the experimental group. To these was sent a somewhat similar letter and an enclosed post card. The letter was as follows:

Dear Mr. Smith,

I am conducting an investigation in collaboration with the Jefferson Medical College to determine the prevalence of sinusitis and hearing loss throughout the country.

I have chosen college graduates for sampling, inasmuch as I think their opinions would be the most accurate.

If you will fill in the return enclosed post card I will greatly appreciate it. Feel free to make any additional comments.

A copy of the published report will be sent to you if you so desire.

Sincerely yours,

Note that in this letter nothing was said about swimming. The return post card was also as similar as possible to the one sent the swimmers. This post card contained spaces for

answers to similar questions as to sinusitis and hearing loss but not mentioning swimming or diving. After each was a space for stating the cause of the trouble if known, a space for comments, name, age and university.

At this time, advice was sought and obtained from The Audience Research Incorporated of Prinecton, N. J., concerning the method of the survey. They kindly furnished a report, the essence of which was that the survey was for the most part being conducted properly, but suggested, among other things, that a repeat letter be sent to a sample of both the experimental and control groups. The purpose of this was to determine whether there was any weighting of the group who did not respond at first. Accordingly some 500 repeat letters were sent to each group. These answers were recorded separately.

It soon became apparent that information other than that obtained from the post card survey would also be desirable. With this in mind, special letters were sent to athletic and public health directors, well known amateur and professional swimmers, and to physicians in charge of college health. Personal interviews were had with several prominent coaches and college authorities, including Mr. Robert Kiphuth, swimming coach, and Drs. Rogers and Philips, student health physicians, of Yale University, Dr. Leroy Mercer, of the University of Pennsylvania, Mr. Howard Stepp and Dr. Raycroft, of Princeton, Mr. Edward Kennedy, of Columbia, and others. All were most cooperative and helpful. In addition a few well known pediatricians were consulted.

Another attempt at gaining information was made by corresponding with doctors, headmasters and coaches connected with boarding schools. This approach was inspired by the work of Dr. Roswell Gallagher,[7] and by the thought that those connected with boarding schools were presumabily more intimate with and hence more cognizant of the boys' activities and illnesses than those connected with the college population.

Finally an effort was made to compare salt water with fresh water swimming. To do this, some of the members of this Society who lived on the seacoasts were asked their opinions on the subject. Many answered this query and in addition some discussed the general subject of swimming in detail. These opinions are included. I express my gratitude to those men without whose comments this paper would lack material of great value.

RESULTS OF THE SURVEY.

There were approvimately 1,800 letters and return post cards sent to the former college swimmers and divers; 410, or 23 per cent, answered. Five hundred repeat letters were then sent, with 123, or 25 per cent, returns.

To the control group were sent 2,500 letters; 613, or 24 per cent, of these answered. Likewise, 500 repeats were sent, with 89, or 18 per cent, returns.

Similar letters were sent to about 170 swimmers, some of whom were professionals and some members of the Amateur Athletic Union; 75 answered.

The total number of form letters with return post cards sent was 5,470, and the total number of replies at the date of writing was 1,310, or 24 per cent. A few more, too late to include, were also received.

Of the 1,310 replies, only those cards, with the exception of a few whose location was not known, which were completely and correctly filled out were included in the results. Those discarded included those who did not give their age or class and those mentioning having one lesion, *e.g.*, sinusitis, but omitting any check mark concerning hearing loss. It could not be assumed that failure to answer meant a negative answer. Due to a mistake, there were too many older people in the control group. Some of these were, therefore, discarded. This was done at random. All over 50 years of age were eliminated, although separate reference is made to this group. With these deletions, the total was reduced from 1,310 to 919: 480 swimmers and divers and 439 controls.

In such a study, certain comparisons present themselves. They are: *1.* comparisons of the experimental with the control group to determine the difference, and to determine if the control group was a satisfactory one, *2.* comparisons of figures within the separate groups, and *3.* comparisons of the combined groups with already known information.

First to be considered was the number of those reporting disease, hereafter referred to as the positives, encountered in each group.

AMOUNT OF DISEASE.

	Experimental	Control
Number in group	480	439
Number positives	213	200
Per cent positives	44	46

The percentage positive in the two groups is almost identical. This was a decided surprise. Also, the large percentage of positives was unexpected. The significance of these results will be discussed later under comments.

As was stated, repeat letters were sent to a number in each group to determine if there was any weighting of those who did not first answer.

REPEATS.

	Experimental			Control		
	No.	No. Pos.	% Pos.	No.	No. Pos.	% Pos.
Originals	359	162	45	363	170	47
Repeats	121	51	42	76	30	40

In both groups, the per cent positive in the repeat groups is slightly less than in the original group, but not large enough to be of significance. When these figures are broken down into individual lesions, *i.e.*, sinusitis and hearing loss, the same

holds. One may conclude that those who did not answer the first letter had neither more nor less sinusitis or hearing loss than those who did.

Next to be considered was the prevalence of each lesion, *i.e.*, sinusitis, tinnitus and hearing loss, both in the experimental and control groups.

KIND OF TROUBLE.

	Experimental	Control
Number in group	480	439
Total sinusitis	174 or 36%	181 or 41%
Total hearing loss	46 or 10%	41 or 9%
Total tinnitus	18 or 4%	15 or 3%
Hearing loss plus tinnitus	64 or 13%	56 or 13%
Both sinusitis and hearing loss or tinnitus	25 or 5%	42 or 10%

Several findings might be noted: *1.* there is a marked similarity between the two groups with every lesion; *2.* the sinusitis figures seem high. The hearing loss in itself is not particularly so, unless combined with the tinnitus; however, it might be a sound assumption that those reporting tinnitus do have a slight hearing loss; *3.* those having both sinusitis and hearing loss or tinnitus are twice as numerous in the control group. This is probably coincidental.

The geographical areas as represented in the two groups are presented. The thought was to compare the two groups and particularly to find if there was any greater tendency to lesions in those living in one or another place. Three locations are shown: *1.* South, which comprises all areas south of the Mason-Dixon line, Louisiana, Arkansas, Oklahoma, Texas, New Mexico, Arizona, Nevada, California and Hawaii; *2.* Philadelphia; and *3.* North. North includes all other parts of this country. Philadelphia was chosen as a particular area because it was thought that a comparison with a large city would be interesting, due to the dust, smoke and dirt present there.

LOCATONS.

1. Comparisons between experimental and control groups.
2. Percentage of positives in various localities.

	Experimental			Control			Total		
	No.	No. Pos.	% Pos.	No.	No. Pos.	% Pos.	No.	No. Pos.	% Pos.
South	97	47	48	152	77	51	249	124	50
Philadelphia	77	42	55	183	79	43	260	121	47
North	296	118	40	99	40	40	395	158	40
No location noted	10			4			14		

Unfortunately, much of the South group came from the border areas, such as Maryland, Virginia, West Virginia and Washington, D. C., and so cannot be regarded as the deep South; however, a more detailed break down, although containing too few cards to be significant, did not show any appreciable difference.

It is evident that the experimental and the control groups were not well selected for geographical similarity, the experimental containing more from the North and the control more from the South and Philadelphia; however, the per cent of positives in each locality is nearly the same for each group.

Also, it is evident that the per cent of positives in each location, regardless of group, is similar. We may conclude, therefore, that the amount of disease shown in this survey does not differ as to locality.

The positives in the experimental group were divided into three subgroups, according to what was thought to be the cause of the trouble. First were those who claimed their trouble was from swimming and diving; second, those who did not know, and third, those who thought the trouble was not due to swimming and diving.

CAUSE OF TROUBLE IN EXPERIMENTAL GROUP.

	Positive 1 Trouble from Swimming and Diving	Positive 2 Don't Know	Positive 3 Not from Swimming and Diving
	No.	No.	No.
Sinusitis	54	35	60
Hearing loss and tinnitus	11	17	11
Both	13	10	2
Total	78 (11m) or 16%	62 (3m) or 13%	73 (4m) or 15%

The number in each of these groups is approximately the same. There are 78, or 16 per cent, of the swimmers and divers who thought the sport was the cause of their sinusitis

or hearing loss, and 62, or 13 per cent, who did not. It seems reasonable to suppose that at least some of these people contracted sinusitis or hearing loss from swimming or diving. Shown in parentheses are the number of lesions checked as marked rather than slight. There are more by far in the first group. This finding is suggestive only. Further analysis of the severity of the trouble will follow.

SEVERITY OF TROUBLE.

	Experimental			
	No.		% of Pos. (213)	% of Whole (480)
Slight	195		92	42
Marked	18	16 sin. 2 h. l. or tin.	8	4

	Control			
	No.		% of Pos. (200)	% of Whole (439)
Slight	163		82	37
Marked	37	35 sin. 2 h. l. and tin.	18	8

There is a much greater proportion of marked trouble in the control group than in the experimental group. These findings are statistically significant and will be discussed later.

As may be recalled, the post card asked whether the person was a swimmer, diver, or both, in an effort to determine if one or the other sport was more responsible for the disease.

SWIMMERS VERSUS DIVERS.

	No.	No. Pos.	% Pos.
Swimmers only	351	141	40
Did both	86	44	51
Divers only	42	27	64

The swimmers had the least and the divers had the most infections. It might be proper to consider the ones who participated in both as diving some, but less than the divers group, and it is seen that their percentage of trouble lies between the others. In other words, the percentage of trouble is proportional to the amount of diving. Such figures, however, are only suggestive and are not statistically significant.

Another question asked was whether the person had continued the activity after his college career. Without resorting to figures, it may be said that there was no difference between those who did and those who did not.

The factor of age must be considered. The following table shows the age comparison between the experimental and the control groups and the percentage of positives in those groups.

AGE AND DISEASE.

Age	Col. 1 No.	2 %	3 No. Pos.	4 % Pos.	5 % of the Age Group Pos.
		Experimental, 480 Total.			
18-30	164	34	72	15	44
31-40	223	46	90	19	40
41-50	93	19	51	11	55
Over 50	6	1	3	1	50
		Controls, 439 Total			
18-30	149	34	65	15	44
31-40	211	48	97	22	46
41-50	79	18	38	9	48
Over 50	65	15			23

Column 2 shows that the percentage of each age in both the experimental and control groups is almost identical. In other words, the two groups were chosen properly in this regard. The age factor, therefore, did not influence the results. As has been stated, the over 50 group was not included except when specifically noted.

In column 4 it is seen that there is a close similarity for each age between the percentage of positives of the whole in the experimental and control groups, *i.e.*, 15 per cent in the 18-30 for both, 19 per cent in the experimental and 22 per cent in the control for 31-40, etc.

Column 5 shows the percentage of positives of those in that age group in each of the groups. There is no significant trend; the per cent positives do not differ as to age.

Age and kind of disease is shown in the table below. It was thought worthwhile to determine whether the separate lesions, *i.e.*, sinusitis or hearing loss, varied according to the age. Both experimental and control groups were combined for this study as the figures of each were similar.

TABLE A.

	No. in Age Group	No. Sinus Lesions	%
18-30	313	122	39
31-40	434	162	37
41-50	172	76	44
Over 50	71	18	25

TABLE B.

	Total No. Lesions	No. Sinus Lesions	%
18-30	155	122	79
31-40	216	162	75
41-50	109	76	69
Over 50	43	18	42

This table (A) does not show any marked trend, unless the over 50 group could be regarded as significant; however, if figured another way (see Table B), the percentage of total number of lesions which are sinus lesions, we find a slight decrease in the sinus lesions through all groups as the age advances. It is not statistically significant.

The next table shows a similar break down of the hearing loss and tinnitus combined.

AGE AND HEARING LOSS.

	No. in Age Group	No. Hearing Loss Lesions	% of Hearing Loss	Total No. Lesions	% of Total
18-30	313	39	11	155	21
31-40	434	54	12	216	25
41-50	172	23	19	109	31
Over 50	71	25	35	43	38

Either way this is figured, *i.e.*, per cent of whole age group or per cent of total number of lesions, we note an increase of hearing loss with advancing age; however, again the differences in the three younger age groups are more suggestive than statistically significant.

Reasons given in the controls as causes of sinusitis under "comments" were interesting. "Climate," usually with the adjective "damp," "humid" or "cold," led by far. This opprobrium was used equally for all localities. Other causes included smoke and smoking, nasal deformities, dust, allergy and swimming. Causes given for hearing loss were too varied to enumerate.

Occasionally there was an amusing reply. A college swimming coach wrote that, to avoid infection, "all swimmers before entering the pool, should dip their fingers into the water and dampen their ear lobes — we have found that it is very successful."

OPINIONS FROM SPECIAL GROUPS.

1. Swimming Coaches and Athletic Directors.

Eleven coaches and directors, all with years of experience, wrote interesting replies concerning swimming and upper respiratory infections. Most (seven) thought that no harm

resulted from swimming and diving; two, that there was less infection among swimmers than in nonswimmers, and one thought there possibly was more infection. They did not differentiate between the effects of swimming and diving; however, more than half qualified their opinions, stating that, in order to avoid infection from swimming and diving, *1.* proper breathing was essential, *2.* there must be proper attention to diet (vitamins, etc.), rest and general hygiene, including avoidance of chilling after leaving the warm gymnasium, and wearing rubbers on wet days. As one said, "I must always keep after the boys." Several mentioned that swimming and diving would aggravate sinusitis if already present, and one conceded that perhaps the lack of infection among the college swimmers was due to the fact that they were pretty well screened out, *i.e.*, only healthy ones swam.

The general trend of their opinions, *i.e.*, the denial of infections from swimming and diving, was most striking. Several experienced coaches stated that they did not recollect ever having had to prohibit a man from entering a meet because of upper respiratory infection. It must be remembered, however, that coaches depend upon the sport for their livelihood and, therefore, might be partial. They also work only with the healthy athlete.

Saunders[8] had some interesting replies to questionnaires answered by 18 swimming instructors attached to various athletic clubs. Most of these felt that there was very little infection among the swimmers and divers, although one or two reported considerable. Saunders, himself, both an expert swimmer and otolaryngologist, thought that most disease was contracted by two groups: beginners, and expert divers and polo players. The former because of improper breathing, and the latter because of the excess strain placed upon their respiratory tracts.

2. College Health Physicians.

College health physicians were almost but not so unanimous or emphatic in their opinions that swimming and diving

caused little trouble. Dr. Raycoft, of Princeton, stated that he has been in close contact with swimmers and divers, including Olympic swimmers, for years, and that he knew of no one seriously inacapacitated from upper respiratory infection or hearing loss. Dr. Leroy Mercer of Pennsylvania agreed. Others felt there was a slight amount of infection incurred.

WATER POLO.

If water sports ever caused infection, it would be in the game of water polo. Here the participants are submerged much of the time and may not have time to breathe properly before submerging. They are also subject while submerged to blows upon the ears. Nearly all consulted admitted occurrences of traumatic nature of the eardrum. Opinion was divided, however, as to the game's being responsible for upper respiratory infection. Several doctors (Roger and Philips) and one well known coach (Stepp) stated that there was some infection caused, while others (Mercer and Kennedy) denied this. One friend of mine, a former player and now a doctor, had to stop because of marked sinusitis resulting directly from it, while another, also experienced and still playing the game, stated he had never contracted an upper respiratory infection. He did, however, admit to several traumatic ruptures of the drums, but attached little significance to them, and said that his hearing was perfect.

Regardless of individual opinions, water polo has been discontinued as a college sport, mainly because of health.

3. Boarding School Personnel.

Dr. Roswell Gallagher,[9] of Phillips Andover Academy, recently studied the incidence of upper respiratory infection among those at his school who did and did not frequent the pool. He found 14 per cent more upper respiratory infections among the former. Many of these, however, were untrained bathers.

Headmasters, coaches or doctors connected with 12 other similar institutions replied. Five of these denied any trouble

from swimming and diving. Two felt there would be no trouble providing the swimmers had a normal upper respiratory tract. One felt that swimming does cause infection, and four would not offer an opinion. In other words, there was a more or less denial of trouble.

4. Pediatricians.

A few well known pediatricians were informally consulted on this problem. They were unanimous that among children the incidence of upper respiratory infections from swimming and diving was noticeable or even marked. The reasons were obvious, and included the anatomy of the child's upper respiratory tract, the playing in water, chilling and general disregard for hygiene so characteristic of children. It is to be remembered that most of the reported cases of severe infection attributable to swimming were among the young.

5. Individual Opinions of Expert Swimmers.

There were many individual opinions from professionals and Amateur Athletic Union members. These letters often were detailed as to the swimming habits of the individual, how to avoid trouble, and his general impression of the sport. The great majority expressed high praise for it. Many stated they had been swimming and diving for years without even any sign of upper respiratory infection attributable to it. Some even claimed water sports prevented infection.

It would seem probable that the more enthusiastic swimmers would be the ones to offer their opinions. This would modify the importance of their statements.

SALT WATER VERSUS FRESH WATER.

While considering swimming in general, a question presented itself which seemed worthwhile investigating. It concerned the difference, if any, between the effects of swimming in salt or in fresh water. By salt water is meant sea water or its equivalent.

Fenton,[10] in 1923, showed there was a destructive action of fresh water on the nasal mucosa which resulted in lowered local resistance to bacteria. As he pointed out, the osmotic action, driving water into the tissues, produced edema, acidosis, loss of ciliary action and eventual cell death. Proetz[11] found that all ciliary action ceased at a concentration of 0.2 or 0.3 per cent salt: the cells became clouded, and that this cessation of motion was permanent. On the other hand, he found the stoppage to occur in hypertonic solutions at 4 to 5 per cent, which is about 1 per cent stronger than ocean water, but in this case the action commenced again if the tissue were immersed in normal saline. Most (75 per cent) of the rabbits investigated by Stark[12] developed purulent sinusitis. Proetz[11] showed that there was also a slowing of the cilia with cold water.

In addition to the local effect on the tissues, there is another factor to be considered. The edema, mentioned by Fenton, which is produced by fresh water, will temporarily at least cause any water and bacteria which have entered the sinuses or middle ear cavities to remain there, producing further local damage and possible pus formation. On the other hand, hypertonic solutions, such as ocean water, will shrink the tissues and allow easy evacuation.

A practical consideration of some importance is the fact that most salt water swimming is done in the open ocean where there is little or no contamination, usually bright sunlight, and plenty of beach to run on to keep warm; however, if there is contamination in salt water, upper respiratory trouble does result, as was stated by Pinkerton.[13] In his locality, sewage from a nearby city will, under certain conditions, reach a popular bathing beach. He noted a definite increase of upper respiratory disease when this occurred.

Twenty otolaryngologists, all members of this Society, were kind enough to reply to my queries. Besides opinions upon the question of salt versus fresh water, other thoughts relating to swimming as a cause of upper respiratory infection were offered.

Eleven of the 20 thought that fresh water swimming was more harmful, while one thought that ocean beach bathing was so, due to the entrance of sand into the nasal passages. Seven thought there was no difference, and one ventured no opinion. Thus there was a predominance of opinion in favor of salt water swimming.

Other factors mentioned leading to upper respiratory infection follow: Heading the list was the contamination of the water. Twelve mentioned this. Infection already present in the upper respiratory tract of the swimmer was mentioned nine times. Diving and under water swimming were next in importance, followed by the presence of irritants, chilling, exposure, exhaustion, and finally improper breathing. Three felt that swimming caused little or no trouble, and one wondered if salt water bathing might not improve some of the chronic nasal conditions.

It is interesting to compare the viewpoint of the otolaryngologist in regard to swimming and upper respiratory infection with that of the swimming coach.

COMMENTS.

The post card survey is open to several justifiable criticisms: *1.* the exact age of the swimmers is not known. The class rather than the age was asked for in the experimental group. The age then had to be computed from the class, assuming that graduation was at age 22; *2.* the polled perhaps did not know what was meant by sinusitis. Sinusitis is a vague term to the layman, and slight sinusitis can be and is claimed by so many that its significance is probably small; *3.* the geographical location of the two groups is not similar. Break down of this, however, does not alter the results; *4.* the sample is a fairly small one, subject to error because of its size; *5.* a control group consisting entirely of athletes would have been preferable to the one used. Finally, whether it be called a criticism or a limitation, the post card survey only embraces the trained adult swimmer, not the child, or the "bather." The names sent by the colleges were those listed

as letter men or at least swimming squad men. To achieve this proficiency in swimming or diving, the person would presumably be one who had not contracted severe upper respiratory disease in the past. Also, when trying out for the team, those with or contracting any marked upper respiratory infection would not be allowed to participate. Then, too, when once accepted as a candidate, the swimming and diving is done under strict supervision. Coaches emphasize their watchdog care. The swimmers and divers are taught to breathe so as to keep as much water as possible out of their noses. They practice a limited time, do not "play" or sit on the cold marble and become chilled.

It is evident, then, that the survey deals with but a small selected group; however, any disease contracted by those in this group would be included in the survey. This covers perhaps one to three years of swimming at college, and all subsequent infection. For such a group, the survey is probably reasonably accurate.

Several of the statistical results need some comment: first is the close similarity between the experimental and the control groups.

It is known that water enters the nasal passages of even the expert swimmer. It is noteworthy, therefore, that so little apparent harm resulted from such frequent immersion, even in this selected group, and may be regarded as a tribute to the coaches and trainers. Perhaps the supposition that athletes enjoy better health than nonathletes, as is claimed by some,[14] is also partly responsible; or it could be said that these findings indicate that athletes do enjoy better health or, more precisely, resist infection more than do nonathletes. The lesser number of severe lesions in the experimental group tended to substantiate this.

Another possible explanation for the similarity of the two groups might be that infection even among bathers is rare and would not appreciably affect statistics. If this be true, one would have to disregard the 78 who stated their trouble came from water sports.

The large percentage of positives in all groups may be due, as has been mentioned, to failure to identify sinusitis. Psychic factors or inclusion of other ailments, such as allergy, also must be considered. The hearing loss plus tinnitus figures (13 per cent) are high and difficult to account for, but hearing loss alone, however (9 to 10 per cent), is only slightly above other estimates.[15]

Also, it is probable that a direct questionnaire such as this would elicit a maximum number of positive responses.

When the number of severe lesions in the experimental group is considered, one notes that there were 11 among the 78 who stated their trouble came from swimming and diving, and only seven among the 125 comprising the rest. This would suggest that lesions among the swimmers due to swimming were more apt to be severe ones.

The opinions of the special groups varied according to the group in question, and these opinions must be weighed in this respect. Coaches see the healthy, trained athlete; otolaryngologists and pediatricians, the sick patient. It is to be remembered that these groups, except for the coaches, are concerned mainly with the great mass of bathers, rather than with the trained swimmers.

CONCLUSIONS.

1. The incidence of sinusitis and hearing loss among healthy adult trained swimmers was no greater than among unselected college graduates.

2. There was less severe upper respiratory infection among the swimmers and divers than among the controls.

3. In the experimental group, the figures suggested that lesions contracted from swimming and diving were more apt to be severe than those contracted from other causes.

4. Geographical location bore no definite relation to the incidence of disease.

5. Coaches and other swimming experts saw little infection; school and college physicians and boarding school personnel saw more; otolaryngologists still more, and pediatricians, most.

6. Salt water swimming was considered safer than fresh water swimming.

7. Factors emphasized leading to the contraction of disease were lack of proper training and supervision, immaturity, the presence of already existing upper respiratory infection, and contaminated water.

SUMMARY.

An attempt was made to determine the relationship of swimming and diving to sinusitis and hearing loss. A letter and post card survey was conducted among some 900 college graduates, roughly half of whom were trained swimmers. Other information was obtained from correspondence and interviews with swimming coaches, school and college personnel, otolaryngologists and pediatricians. The relative merits of fresh versus salt water swimming were discussed.

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12. Stark, W. B.: Irrigations with Aqueas Solution: Their Effect on the Membranes of the Upper Respiratory Tract of the Rabbit. *Arch. Otolaryngol.*, 8:47, July, 1928.

13. Pinkerton, F. J.: Personal communication.

14. Gallagher, J. R., and Brouha, L.: Physical Fitness: Its Evaluation and Significance. *Jour. A. M. A.*, 125:834, July 22, 1944.

15. Macfarlan, Douglas: Personal communication.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The coming Section Meetings of the American Laryngological, Rhinological and Otological Society, Inc., are to be held as follows:

Eastern Section: Hotel Statler, New York, N. Y., Jan. 5, 1950.

Council Meeting: Hotel Statler, New York, N. Y., Jan. 6, 1950.

Combined Meeting, Southern and Middle Sections: Peabody Hotel, Memphis, Tenn., Jan. 16-17, 1950.

Western Section: Medical Society Building, Los Angeles, Calif., Jan. 21-22, 1950.

The 1950 Spring Meetings will be held at the Hotel Mark Hopkins in San Francisco, Calif., as follows:

American Board of Otolaryngology, May 16-20, inclusive.

American Otological Society, Inc., May 21-22.

American Laryngological Association, May 23-24.

The American Laryngological, Rhinological and Otological Society, Inc., May 25-27, A.M.

The American Broncho-Esophagological Association, May 25-26, P.M.

Please make your Hotel Mark Hopkins reservations at once by writing to Mr. Carl J. Wilson, Assistant Manager, San Francisco Convention and Tourist Bureau, 200 Civic Auditorium, San Francisco, Calif., and enclose a deposit of $10 made payable to the Hotel Mark Hopkins. In return you will receive a hotel reservation confirmation. The limited facilities available make early reservations important.

Members and guests desiring to go on special Pullman cars "with the gang" should write immediately to Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill., Chairman of the Transportation Committee.

Dr. C. Stewart Nash, Secretary.

SECOND PAN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY — MONTEVIDEO-BUENOS AIRES, JAN. 8-15, 1950.

EXECUTIVE COMMITTEE.

President: Prof. Justo M. Alonso.

Vice-Presidents: Prof. Elias Regules, Dr. Juan Carlos Munyo, Prof. Juan Carlos Oreggia.

Secretary General: Prof. Pedro Regules.

Secretary of Interior: Prof. Hector Rebagliatti.

Secretary of Exterior: Dr. Julio Cesar Barani.

Secretary of Exhibit: Dr. Enrique Apolo.

Secretary of Publicity: Dr. Jaime Sala López.

Treasurer: Dr. Alberto Santoro Vecino.

Assistant Treasurer: Dr. Juan Carlos Munyo, Jr.

Delegate in Argentine Republic: Prof. Juan Manuel Tato.

Delegate in México: Dr. Ricardo Tapia Acuña.

Delegate in Cuba: Dr. Pedro Hernández Gonzalo.

Delegate in the U. S. A. and Canada: Dr. Chevalier L. Jackson.

An interesting and instructive program has been arranged.

Subscription—$10, or its equivalent in Uruguayan money.

The following week (Jan. 16-21) there will take place in Montevideo and Buenos Aires, operative demonstrations for those who wish to attend.

Travel arrangements should be made immediately through Thomas Cook & Sons or Pan American Airways.

All those interested in attending this Congress should communicate at once with the delegate in the U. S. A. and Canada, Dr. Chevalier L. Jackson, 3401 North Broad Street, Philadelphia 40, Pa.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT ST. LOUIS, SEPT. 28, 29, 30, 1949.

The Fourteenth Annual Meeting of the Mississippi Valley Medical Society will be held at the Jefferson Hotel, St. Louis, Mo., Sept. 28, 29, 30, 1949, under the presidency of Dr. Alphonse McMahon, of St. Louis University. At the recent current meetings of the Society and board of directors, the following officers were elected: Dr. Nathaniel G. Alcock, Iowa City, Iowa, President-elect; Dr. Wendell G. Scott, St. Louis, Mo., First Vice-President; Dr. Charles F. Harmon, Springfield, Ill.; Second Vice-President; Dr. John I. Marker, Davenport, Iowa, Third Vice-President; Dr. Harold Swanberg, Quincy, Ill., Secretary-Treasurer; Dr. Ralph McReynolds, Quincy, Ill., Accounting Officer.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1949.

Acousticon Model A-100.
Manufacturer: Dictograph Products Corp., 580 Fifth Ave., New York 19, N. Y.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Precision Table Hearing Aid.

Manufacturer: Precision Electronics Co., 850 W. Oakdale, Chicago 14, Ill.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone; Radioear Permo-Magnetic Multipower—report not yet published.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Ravox (Semi-Portable).

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Ralph A. Fenton, 906 Medical Arts Bldg., Portland, Ore.
Secretary: Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

Eastern Section, Hotel Statler, New York, N. Y., Jan. 5, 1950.
Council Meeting, Hotel Statler, New York City, Jan. 6, 1950.
Combined Meeting, Southern and Middle Sections, Peabody Hotel, Memphis, Tenn., Jan. 16-17, 1950.
Western Section, Medical Society Bulding, Los Angeles, Calif., Jan. 21-22, 1950.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Conrad Behrens, 35 E. 70th St., New York, N. Y.
President-Elect: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9-14, 1949.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Rea E. Ashley, 384 Post St., San Francisco, Calif.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22 Pa.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.
Second Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology: Montevideo, January, 1950

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Warren A. Wilson.
Secretary-Treasurer: Dr. Victor Goodhill.
Chairman of Section on Ophthalmology: Dr. George Landegger.
Secretary of Section on Ophthalmology: Dr. Harold B. Alexander.
Chairman of Section on Otolaryngology: Dr. Alden H. Miller.
Secretary of Section on Otolaryngology: Dr. Leland R. House.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Alfred Schattner, 115 E. 61st Street, New York 21, N. Y.
Secretary: Dr. Norman N. Smith, 291 Whitney Avenue, New Haven 11, Conn.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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President: Dr. M. Valentine Miller, 114 W. Phil-Ellen St., Philadelphia, Pa.

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Tesorero: Dr. Alfredo M. Petit.

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ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Joao Penido Burnier.

First Secretary: Dr. Gabriel Porto.

Second Secretary: Dr. Roberto Barbosa.

Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.

Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.

Vice-Presidente: Dr. Luis E. Olsen.

Secretario: Dr. Eugenio Romero Diaz.

Tesorero: Dr. Juan Manuel Pradales.

Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.